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DISEASES OF INFANCY AND CHILDHOOD

BY

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With a Foreword by the late
Sir FREDERIC STILL, K.C.V.O.
M.A., M.D., LL.D., F.R.C.P.

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FOREWORD

It is pleasant to wish " bon voyage " to a good ship starting on its travels, though we be but lookers-on as she slips her cable and sets out we know not whither. Of one thing I feel sure, that this book with its freight of valuables will travel far.

Having been, as it were, allowed a private view, I know that it carries not merely the stock-in-trade of every medical textbook, but the fruit of personal experience, so that opinions expressed have been checked by clinical observation, and as such are of solid worth.

There was room for yet another book on diseases of children, for the progress of knowledge in this particular department of medicine has been rapid during the past few years. There is for Medicine no " abiding in one stay " and we need to be kept up to date constantly. It is the function of such a book as this to keep us abreast of recent developments and particularly of the latest methods available for helping children in their time of sickness ; in this Dr. Sheldon's book will do good service and one can heartily wish it success.

FREDERIC STILL.

London,
July, 1936.

PREFACE TO THE FOURTH EDITION

It is with a sense of personal loss that the death of Sir Frederic Still on June 28th, 1941, must be here recorded. It was the author's privilege to work with, and learn from, Dr. Still (as he then was) for more than ten years ; much of his teaching, and a little of his vast knowledge of children and their ailments, find expression between the covers of this book, and if this book could contribute in some small way to the survival of his influence for the welfare of children in their time of sickness, the author would indeed hold himself fortunate. Sir Frederic Still was for many years the acknowledged leader of Pædiatrics in this country, and with his death British Medicine has lost a great physician.

In spite of the War, the study of children's disease has advanced, calling for revisions and additions in the preparation of this edition. The chapters on prematurity and diseases of the new-born have particularly felt this impact. Although the sulphanilamide drugs find increasing mention, new members of this group are constantly being produced, and therefore recommendations for the use of individual preparations in particular conditions have in general been avoided. The subject of undescended testicle, as it appears to the physician, has been discussed. In the account of Scarlet Fever, the relationship between this disease and hæmolytic streptococcal tonsillitis has been amplified, revealing a view divergent from Public Health practice. Of war-time measures, national dried milk, cod liver oil compound, black currant purée, and rose hip syrup have required inclusion. New treatments include benzyl benzoate in scabies infection, spinal anæsthesia in Hirschsprung's disease, Atebrin in Lambliasis, and notes on Rhesus negative blood transfusion in icterus gravis and ligation of patent ductus arteriosus.

The number of prescriptions has been slightly reduced, and those that remain have been written in full. A plate of cystic disease of the lung, and three illustrations showing œsophageal defects and tuberculous obstructive emphysema have been added.

WILFRID SHELDON.

PREFACE TO THE FIRST EDITION

When I was approached by Messrs. J. & A. Churchill some eight years ago with the suggestion that I should write a book dealing with the diseases of children, it was not without diffidence that I accepted, for at that time there seemed so many books concerned with the subject. I decided that it should be something more than a mere handbook, and yet in no sense competitive with the larger works of reference with which the medical libraries are well stocked. Its contents represent the clinical teaching and lectures on pædiatrics given in the Children's Department, and in the post-graduate courses, at King's College Hospital. I venture to hope that the book may prove useful to a wider circle of both undergraduate students and doctors in practice.

During the course of preparing the book, I have derived both pleasure and profit from collecting the necessary material, and from sifting the views of modern writers while at the same time holding fast to the wisdom, born of their long experience, of my own teachers and their contemporaries. With regard to the subject-matter, I have not confined myself merely to matters of diagnosis and treatment, for these are aspects of medicine which can only be viewed in their proper perspective against a background of etiology and pathology, and the study of disease would be lacking in balance if only the foreground were showing. I have attempted to give to the common diseases the space which is their due, seeing that they make up the bulk of practice, but I have not hesitated to include rare diseases as well, for we never know when we may meet them, and their recognition is an unfailing source of stimulation and satisfaction.

I have found some difficulty in deciding which of the surgical conditions should be included and to what extent comment should be made upon them. My object has been simply to represent them as they come before the physician. Conditions such as tuberculosis of bones and joints, osteomyelitis, and cleft-palate, for descriptions of which one would naturally turn to a surgical textbook, have been intentionally omitted.

My record of indebtedness is a long one. First and foremost is my gratitude to my teachers for the information which I have consciously and unconsciously absorbed from them, and whose views I, at least, can recognise in the pages that follow ; in particular to Dr. Still, under whom, as clinical clerk, house-physician, registrar, and eventually junior colleague, it was my privilege to serve. It is fitting also to acknowledge my debt to those who, during the last eighty years, have compiled the store of information which reposes in the records of The Hospital for Sick Children, and in the Hospital's museum. I also acknowledge with pleasure the help of Dr. McCance, Biochemist to King's College Hospital, who kindly read through the sections dealing with nephritis and the disorders of metabolism ; and to Mr. James Crooks, Aural Surgeon to The Hospital for Sick Children, Great Ormond Street, who dealt in a similar way with the chapter on affections of the ear, nose and throat, and in addition wrote for me the short section on the nasal accessory sinuses. I wish to thank Mr. Deryck Martin, and Miss Hunt,, who have toiled for me in photographing my cases at Great Ormond Street and King's College Hospital respectively ; and to thank the Editors of the *Lancet*, *Archives of Diseases in Childhood*, *Quarterly Journal of Medicine*, the *Practitioner*, and the *Australian Medical Journal*, for allowing me to reproduce portions of contributions which I have made to these journals.

The list would be incomplete if I failed to acknowledge to my Wife her encouragement, and abiding patience, through the years that have gone to the making of this book.

In conclusion, I wish to record my appreciation of the helpful advice and courtesy at all times of the Publishers, and of their patience with an author who far exceeded the time allotted for the completion of his book.

WILFRID SHELDON.

31 Wimpole Street, W.1

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DISEASES OF INFANCY AND CHILDHOOD

CHAPTER I

ON THE EXAMINATION OF CHILDREN

THE medical curriculum is generally so arranged that the student does not begin the special study of children until he has already received instruction in the examination of adults, and has been taught that the signs of disease are to be elicited by the processes of inspection, palpation, percussion, and auscultation, carrying them out in a methodical order, and applying them to each system of the body in turn. These methods, coupled with a careful history, are the foundations on which his diagnosis must be built.

In older children it is usually possible to follow the same routine, but in infants and young children two difficulties become at once apparent. In the first place the young child is incapable of describing his symptoms. and for the history of the illness one has to rely on the hearsay evidence of the parents, whose testimony, be it said, is usually invaluable, but like that of other witnesses, may be both biased and misleading. In the second place a methodical examination calls for co-operation on the part of the patient, and with a child this is not always forthcoming. With regard to the order in which the examination shall be conducted it is the child who calls the tune, and the doctor must be prepared to vary his routine to suit the whims of his small patient. On this account it is all the more easy to overlook various details of the examination, omissions against which the doctor must be constantly on his guard. In particular the examination of the urine, the ears, and the tonsils, are easily forgotten, the urine perhaps because of the alleged difficulty of obtaining a specimen, the tonsils because of the known objection of many children to having their throats inspected. Another reason for overlooking disease in these situations is that the symptoms are often of the vaguest, and there may be no hint at all that the cause, perhaps of a high temperature, or of sickness, or of a convulsion, lies in infection of the kidneys or in otitis or tonsillitis. Therefore it should be a maxim that when the cause of a child's illness is not satisfactorily explained, a specimen of urine must be examined (the methods of collection are mentioned in the appropriate chapter), and the ears and throat must be inspected. So that the rest of the examination may be conducted peacefully, inspection of the throat should be left to the last—last, but very far from least in importance.

A careful history is never more necessary than when the subsequent examination is likely to be difficult, and therefore in children it is of the utmost importance to have all the data leading up to the present condition clearly before one, indeed it is often the case that quite as much time must be spent on questioning the parents as on examining the child, for there is hardly anything more likely to undermine one's confidence when making the examination than to have to go to work with a muddled and disjointed history. There are many points in a child's history which would be of no importance in later life. For instance there are the details of the pregnancy and birth; whether the mother was healthy throughout her pregnancy, and whether the child was born at full term, whether the labour was normal, what was the weight at birth, and whether the cry of the new-born child was vigorous, and breathing and sucking were easily established. Cerebral damage sustained at birth almost always interferes with these functions.

During infancy the details of the diet must, of course, be gone into very fully, and in older children also it is as well to know the sort of routine by which the child passes his day, and also to enquire into the foods he is having, for herein may lie the explanation of such everyday complaints as loss of appetite, irritability, fatigue, unpleasant breath, or the passage of unhealthy or slimy motions. The physical and mental growth must also be gauged by enquiring whether the various milestones of development have been reached at the appropriate age, for instance, an infant should be able to raise the head off the pillow at three months, should be sitting up by nine months, and should be walking by eighteen months. Talking, to the extent of stringing two or three words together, should be acquired by two years, and most infants by this time have also learned to control their sphincters.

The family history is particularly important in children, partly because inherited abnormalities are so likely to make their appearance in childhood, and also because children are peculiarly susceptible to infective conditions in their environment. To take an example, the knowledge that there is in the home a phthisical adult will at once be a warning not to dismiss lightly a child's cough or his failure to thrive until it has been established by every means available that the child is free of tuberculous infection. We must also know where the child comes in the family order, the health of his brothers and sisters, and whether there have been any premature births, still-births, or miscarriages.

Questions must also be asked about any previous illnesses. Speaking generally, the infectious fevers seldom occur twice, and therefore if one is doubtful whether a child has, say, chicken pox or mumps, a clear story of a previous attack would be valuable evidence against the diagnosis. We know also that broncho-pneumonia, especially if it complicates measles or whooping-cough, is very likely to go on to pulmonary fibrosis, and a history of these illnesses may suggest the explanation of a recurring winter cough. The relationship between eczema in infancy and asthma in older children, between bilious attacks and migraine, and between chorea, rheumatic fever, and rheumatic heart disease, afford further examples of the importance of knowing a child's past illnesses.

Lastly there follows the history of the complaint in question, which must be ascertained in the same way as in adults. The exact time and mode of onset is often illuminating, for instance, when abdominal pain and vomiting have occurred it is essential to know which came first, for this may have considerable bearing in distinguishing between conditions such as appendicitis and a bilious attack. The value of a precise history is also illustrated by asthma, for the account of the attack may indicate at once whether one is dealing with true spasmodic asthma or with asthmatic bronchitis, the management of the two conditions being along quite different lines. Another symptom much more common in children than in adults is a fit, and unless the history is carefully taken it is easy to mistake for an epileptic convulsion other attacks such as crying convulsions, masturbation, or fainting.

Turning now to the actual examination of the child, there is hardly need to say that tact and patience are two essential qualities. Some doctors are inherently more successful in handling children than are others, but with practice and a real attempt to see the examination as the child sees it, we shall each of us discover personal ways and means and learn for ourselves various Do's and Don'ts which will make the examination more successful. Should the child happen to be asleep as much of the examination as possible should be made before he wakes. The respiration and pulse rates should be counted, the heart may be auscultated, and, with a warm hand, the abdomen can often be palpated. Even the discs of a sleeping infant can be examined without waking him. In nervous children, when once examination is under way, a gently spoken and friendly " running commentary " helps to keep the tears at bay.

The particular points in each system of the body to which attention must be paid are indicated in the chapters that follow, but I shall make here, for what they are worth, some general remarks about the handling of children which I have personally found helpful. In the first place the less of the armamentarium of one's profession that is showing in the consulting room the better, for glass-topped trolleys and racks of glittering instruments are not likely to ease a child's apprehensions. Then, when the child first enters, leave him to his own devices, let the parents take the centre of the stage, and above all don't stare at the child, for he is not used to it and it is disconcerting to him. Much can be learned from quietly watching him after his attention has been attracted by toys or picture books. Then, when the history has been well and truly taken, the child must be undressed. Leave this entirely to the parents, for the child will more often than not resent any assistance you may offer. Everything down to the chest must come off, for examination is made needlessly difficult if the child is all the time tugging to pull down his vest, while, once the clothes are right off, even the most refractory little person will usually stop struggling, in fact the more difficult the child the more need is there to insist on the clothes being completely removed.

The actual examination may well begin by taking the child's hand, and incidentally noting the presence or absence of finger clubbing, and the shape and length of the fingers. Then, if the patient is an infant, examine the fontanelle, for this is the equivalent of the pulse in adults. Note its size, whether it is depressed (as in diarrhœa) or bulging and tense (indicating a raised intracranial pressure), remembering that in health the fontanelle pulsates in time with the heartbeat. Next turn to the skin, picking it up to see whether it springs back into position as it should do in health. The elasticity of the skin is diminished in conditions of dehydration. A note will meanwhile have been made of the facies, the complexion, the position of the child, and the general state of nutrition.

The examination of the chest and abdomen follows much the same lines as in adults. As in all branches of clinical medicine, *observation* is by far the most valuable means of examination at our disposal, and never more so than in children, for palpation, percussion, and auscultation may all be resisted, but the child cannot deny us inspection. Percussion, to be of any value, must be light, otherwise the more subtle changes in the note cannot be appreciated, and, too, if it is done heavily or clumsily it may soon bring tears. Percussion is seldom of much use in a crying child, for the note then becomes very variable, in fact it is often possible to demonstrate a "cracked-pot" sound over the upper lobes. Auscultation, on the other hand, is not affected by crying, indeed breathing is then deep enough for the entry of air to be easily heard, and at the same time the voice sounds can be tested. Speaking generally, auscultation gives more information in children than does percussion. It is not out of place to add a warning against making a sudden approach with the stethoscope, which may be a strange instrument to the child, and also against darting the stethoscope from point to point over the chest, let it rather slide over the skin. The same holds true of palpating the abdomen, for sudden movements will quickly make the abdomen taut, while if the hand glides gently from place to place the child will remain relaxed. There is little room for routine in examining the abdomen ; after inspection, one naturally begins by palpating in the quadrant opposite to where any tenderness is expected, leaving the painful area until the last. With experience one's fingers gradually acquire a certain deftness, whether the child happens to be lying on his back, wriggling over on his face, sitting up, or even crawling about on the floor. It should be remembered that the play of emotions on a child's face is more vivid then than at any other age, and valuable information is sometimes to be obtained by watching the expression while the abdomen is being felt, for when a tender area is touched, be it over an inflamed appendix, over an intussusception, or in the loin over a kidney the seat of acute pyelonephritis, the face may be seen to pucker or wince.

A complete examination of the nervous system calls for so much co-operation by the patient that it can seldom be so fully carried out in children as in adults. The motor side is, of course, more easily managed than the sensory. A degree of spasticity of the limbs in the newborn is normal, and the plantar response does not become genuinely flexor until well into the second year. Before this age, however, it is usually possible to tell whether the extensor response has any pathological significance, for in the normal infant the big toe flicks quickly up and down, while if the reflex is pathological, the big toe rises more slowly and the other toes spread out fanwise. With patience, and if the room be a little darkened, a satisfactory view of the discs can as a rule be obtained without resorting to mydriatics.

The clinical survey concludes with an examination of the ears, the teeth and pharynx, the urine, and the stools.

Normal Development. Seeing that childhood is a time of continual change and development, it is necessary to know something of the average rate of progress of normal children. Some of the landmarks have already been mentioned. Two standards commonly used are the weight and height in proportion to the age, although it may be pointed out that after about the fifth year children begin to exhibit the physical standards of the stock from which they are sprung, being perhaps short and stocky, or tall, and therefore it is as important to compare the weight and height in relation to each other as to consider them both in proportion to the age.

The following table, compiled from Holt's figures, shows the average weight and height of children from birth up to twelve years. It is useful to be able to commit this table to memory, for parents are always anxious to know how their child's height and weight compare with the normal standard.

Age.	Weight in lbs.	Height in inches.
Birth	7	20
1 year	21	29
2 years	28	33
3 „	33	37
4 „	37	40
5 „	41	42
6 „	45	44
7 „	49	46
8 „	55	48
9 „	61	50
10 „	67	52
11 „	73	54
12 „	79	56

With regard to the weight it is easy to remember 33 lbs. at three years and 49 lbs. (7 × 7) at seven years. At birth, one year, and two years, the weight runs in multiples of 7, while between three and seven years we add on 4 lbs. per year, which takes us exactly to 49 lbs. The increase in weight after seven is more variable, but an addition of 6 lbs. per year is a fair average.

With regard to the height it will be noticed that the length at birth is 20 inches and that this is doubled at four years of age. From then on the child grows 2 inches per year up to twelve years of age. It may also be pointed out that the height in inches exceeds the weight in lbs. until five years of age, when the weight overtakes the height, and from then on the weight in lbs. steadily draws away from the height in inches.

There remain one or two facts concerning growth which should be remembered, for instance that the anterior fontanelle normally closes at about eighteen months, and that whereas the first tooth to erupt in the first dentition is usually a lower central incisor at six months, in the second dentition the pride of place goes to the first molar, which erupts between five and six years of age —hence the term " six-year-old molar ". The rate at which the maximum circumference of the head increases must also be borne in mind, for when the body is small the head may look out of proportion although by measurement it may really be normal, and further, there are certain types of mental deficiency, such as microcephalic idiocy and Mongolian imbecility, which are always associated with a small head, and many other mentally defective children have a head smaller than normal, while on the other hand the milder degrees of hydrocephalus can only be confirmed if the normal measurements are known, and a large head is also common in rickets and achondroplasia. The maximum circumference of the head should increase at the following rate :—

At birth	13 inches
At six months	16 ,,
At one year	18 ,,
At three years	19 ,,
At seven years	20 ,,
At twelve years.	21 ,,

Lastly, when growth is persistently stunted, as in some of the disorders of the endocrine glands, and also when the contrary holds true and the child seems much too tall, the abnormal skeletal growth may be reflected in a too early, or a retarded, appearance of the various ossific centres. The following table has therefore been compiled to show the age at which some of the centres of ossification should appear. The wrist is particularly useful in this respect, for it will be seen that a fresh carpal centre is added in each of the first seven years except the fourth, while the pisiform centre appears at the twelfth year.

Order of appearance of some of the secondary centres of ossification

Age.	Carpus.	Other Centres.
1st year	Os Magnum	Head of femur.
2nd ,,	Unciform	Lower epiphysis of radius. ,, ,, ,, tibia. ,, ,, ,, fibula.
3rd ,,	Cuneiform	Patella ; one centre in head of humerus.
4th ,,		Lower epiphysis of ulna ; Upper epiphysis of fibula ; Great trochanter.
5th ,,	Trapezium Semilunar	Upper epiphysis of radius. Head of humerus now shows three centres.
6th ,,	Scaphoid	Centres in head of humerus coalesce.
7th ,,	Trapezoid	
8th ,,		
9th ,,		
10th ,,		Upper epiphysis of ulna ; tuberosity of os calcis.
11th ,,		
12th ,,	Pisiform	

CHAPTER II

SOME AFFECTIONS OF THE NEWBORN

Introduction. At the moment of birth the infant is suddenly faced with an entirely new environment, to which a rapid adaptation is essential. The establishment of respiration is an urgent matter, and alterations in the path of the circulation and in the components of the blood soon take place. A new method of obtaining nourishment must be set going ; contact with organisms is inevitable ; and the heat-regulating mechanism is called into play, and clearly at first cannot be entirely relied upon, for short exposures of the newborn child either to cold or to warmth will cause the temperature to fluctuate widely. Also at this age. although clinical examination of the infant is conducted along the lines customary for older children, the standard of health has to be estimated by such special indications as the strength of the cry, the power of sucking, and the degree of drowsiness.

During the first week it is not uncommon for the temperature to rise to 101° F. or higher. This has received the name *Inanition Fever*, but is due in all probability to loss of fluid from the body, and a better name for it is *Dehydration Fever of the Newborn.* Tyson, in a study of 1,972 newborn infants, found that dehydration fever occurred in 10 per cent. The loss of fluid is roughly proportional to the drop in bodyweight which all infants sustain in their first week, and it has been shown that the greater the drop in weight the more likely is this form of fever to develop. The prognosis is perfectly good, for the temperature rapidly subsides as soon as sufficient fluid is supplied. This will obtain when the infant begins to draw fluid from the mother's breasts, but meanwhile the fever can be prevented by giving glucose water until lactation sets in.

It remains to point out that fever during the first week may be due to more serious causes, such as intracranial hæmorrhage, sepsis, and pyelitis, but in these conditions the infant will appear obviously ill, whereas dehydration fever produces little disturbance beyond slight restlessness, and is probably often overlooked.

Another condition which occurs in about 50 per cent. of infants towards the end of the first week is swelling of the breasts, accompanied by the secretion of a fluid resembling colostrum. Both male and female infants are affected, and the swelling may last for two or three weeks. It is due to the infant obtaining from the mother just before birth some of the hormone which stimulates her own breasts to secrete. The condition does not call for treatment unless the swollen glands become infected, in which event an abscess is likely to form, requiring incision and drainage.

Asphyxia Neonatorum

Precisely what causes an infant to take its first breath has been much debated, but the recent investigations of Barcroft[1] indicate that lack of oxygen is the exciting stimulus rather than accumulation of carbon dioxide, and, as he aptly expresses it, "from the point of view of the fœtus the gasp is an index pointing, not to life, but to death, and we are faced with the remarkable paradox that the dying gasp of the fœtus is the earnest of life to the individual." The lungs at the time of birth are airless, solid, and sink when placed in water—a condition of *atelectasis*. Complete expansion is not obtained at the first breath, in fact the lungs continue to expand for a few days, and full expansion will take longer in a premature baby or in one who is breathing feebly than in a lusty full-term infant.

The causes of persistent atelectasis and of cyanosis in the newborn fall roughly under two headings :—

(1) Obstruction to the airway, as for instance by inspired mucus, meconium, and liquor amnii, or possibly by undue adherence of the walls of the pulmonary alveoli, or by direct compression by a large thymus. Congenital deformities of the heart may also be a cause.

(2) Feebleness of the respiratory centres, due to immaturity, or to damage by cerebral œdema or hæmorrhage produced during a prolonged and difficult labour.

The degree of atelectasis varies. Complete non-expansion is of course incompatible with life, in less severe cases the whole of a lobe may remain airless, or more usually there are several atelectatic patches scattered through both lungs. Unless aeration of these soon takes place infection is likely to occur, leading to broncho-pneumonia.

The symptoms are more prominent than the physical signs. Cyanosis is present from birth, and at first the infant may appear livid, or in the more severe cases in which circulatory failure and shock have occurred the colour may be pallid, hence the customary division of asphyxia neonatorum into two classes—livida and pallida—the latter bearing the worse prognosis. The one is, however, merely a stage of the other, and the same treatment should be applied to both. The cyanosis may persist, or may recur from time to time, coinciding with periods of apnœa which are followed by a few gasping respirations, or between attacks the respirations may be so shallow that it is difficult to be sure the infant is breathing. The cry is very feeble, the pulse is rapid, and the temperature may be either raised or subnormal.

---

[1] Sir Joseph Barcroft, *Lancet*, 1935 *ii.*, 647.

Examination of the chest may show but little. Expansion is poor, and there may be some indrawing of the lower intercostal spaces. The percussion note is only impaired if a large area of lung remains airless. The air entry is defective, but the breath sounds are usually vesicular. The most characteristic sign is the presence of fine crackling crepitations heard when the infant can be stimulated to take a deep breath. Should only one lung be affected, it may be possible to detect a displacement of the heart towards that side.

The outlook varies. Prematurity, persistent or recurrent cyanosis, the presence of intracranial hæmorrhage, or the association of congenital morbus cordis make the prognosis bad. On the other hand, if the condition is due to obstruction of the upper air passages and these can be cleared, and if the infant's strength can be maintained, recovery may be expected.

Treatment. The three principal measures are to keep the infant warm, to exercise great gentleness in handling, and to maintain a free airway. The treatment of intracranial birth injury is dealt with in the next section.

Vigorous methods of carrying out artificial respiration in the newborn are likely to do much more harm than good. It is first of all essential to clear away any mucus from the mouth and pharynx, and this may be done with a piece of gauze wound round the little finger, or by using some sort of " sucker," such as the rubber bulb of an auroscope. The tongue should then be held forward to open up the airway and to stimulate breathing, and this may be combined with gentle and slow rhythmic compression of the lower part of the chest. Inhalations of oxygen (95 per cent.) and carbon dioxide (5 per cent.) are valuable, the gases being bubbled through warm water and given through a small catheter passed to the back of the nose.

In the event of collapse, the infant should be placed in a warm bath at a temperature of 105° F., to which a little mustard may be added, in the proportion of a tablespoonful to the gallon. If with these measures respiration is not satisfactorily established, $\frac{1}{4}$ c.c. of pituitary extract, $\frac{1}{2}$ c.c. of coramine, or 1 c.c. (gr. $\frac{1}{20}$th) of lobeline hydrochloride may be injected intramuscularly.

Arrested Respiration in the Newborn

Under this title Still [1] has described attacks of sudden apnœa in infants up to a month old who have not previously shown any abnormality of their respiration. The first attack may be fatal, or if this is survived there may be further attacks which are equally dangerous. Prompt artificial respiration is necessary to restart breathing, and a close watch must be kept day and night for at least a week after the last attack before the infant can be considered to be out of danger of a relapse.

The cause is unknown. Immaturity of the respiratory centre has been suggested, but post-mortem examinations have not thrown any light on the condition.

Acute Hæmorrhagic Pneumonia of Infants [2]

This is a rare but fatal condition affecting infants of from a few hours up to a month old. The symptoms consist of rapid pallor, the appearance of a little blood-stained froth in the mouth, and a speedy death. Autopsy shows extensive areas of lung solid with blood. The condition has been supposed to be the result of infection, or may be allied to other hæmorrhagic crises in the newborn such as hæmatemesis and melæna.

OBSTETRICAL INJURIES

Cephalhæmatoma

This consists of an effusion of blood between the skull and the pericranium, and is more likely to occur in vertex than in breech presentations. The effusion gives rise to a soft fluctuant tumour situated over one or other of the parietal bones or over the occiput. It is bounded by the sutures between the skull bones, which distinguishes it from a caput succedaneum and from a meningocele. Rarely there may be more than one tumour. After a few days the edge of the cephalhæmatoma forms a hard ring due to ossification at the line where the pericranium has been raised from the skull, and this may spread until the tumour is encapsuled in a shell of bone, while the skull at the centre of the hæmatoma may become rarefied.

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[1] Still, G. F., *Lancet*, 1923, *i.*, 431.

[2] Browne, F. J., *Brit. Med. Jour.*, 1921, *ii.*, 144.

As a rule no treatment is required, although if the swelling is unsightly because of its size, with strict asepsis some of the blood can be easily aspirated. Depending on its size and the degree of ossification which takes place, the tumour may take several weeks or months to absorb, but it eventually does so and the contour of the skull becomes normal.

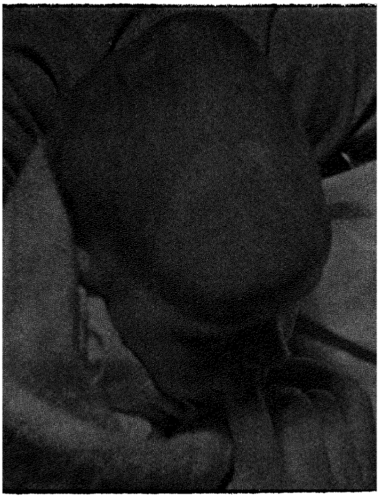

FIG. 1. Cephalhæmatoma over the left parietal bone, in an infant one month old.

Intracranial Hæmorrhage

Intracranial hæmorrhage produced at birth may be situated between the skull and the dura mater (internal cephalhæmatoma), between the meninges, or in the substance of the brain. Of the various predisposing factors, prematurity, difficult vertex presentations, breech presentations, and precipitate labour are the most important. In his enquiry into the causation of fœtal deaths, Holland showed the importance of tears of the tentorium cerebelli, which cause hæmorrhage through rupture of the vein of Galen or its tributaries.

It sometimes happens that the newborn infant presents symptoms suggesting concussion, but under treatment recovers completely in a few days, and in such cases it seems likely enough that cerebral œdema has occurred rather than actual hæmorrhage.

Symptoms. The symptoms date as a rule from birth, but may be deferred for as long as a week. When the injury has been severe the infant is likely to be born in a state of asphyxia pallida, and survival may be only a matter of minutes or hours, or there may be considerable difficulty in establishing respiration, the breathing is shallow, and there may be periods of apnœa with a return of cyanosis. The cry is feeble, indeed the very quietness of the baby may be most sinister, the power of sucking is greatly interfered with, and the infant may seem unable to swallow. The muscles are hypertonic, the limbs are rigid, the reflexes may be exaggerated, and the infant may lie in an opisthotonic attitude. Sudden starts are common, and convulsions, either single or numerous and generalised or Jacksonian, may occur. The anterior fontanelle is sometimes visibly distended and tense, the pupils are usually small and may fail to react, and nystagmus, squints, and local paralyses may be present. The cerebro-spinal fluid may be diffusely blood-stained or show a yellow discoloration, but a normal fluid does not disprove the presence of hæmorrhage. Caution must be exercised in drawing off the fluid, for there is a risk of the cerebellum and medulla becoming compressed in the foramen magnum, and therefore the fluid must be removed slowly, and only a small amount should be allowed to escape.

Prognosis. This depends upon the site and extent of the hæmorrhage. Bleeding into the posterior fossa is, as a rule, rapidly fatal. In other situations recovery may be complete, but permanent sequelæ are likely, their character depending on the position of the hæmorrhage. The more important include mental defect, spastic paralysis, athetosis, convulsions, and hydrocephalus, or a mixture of these.

Treatment. The infant should be nursed in a darkened room in quiet surroundings. It is of the utmost importance to keep him warm. Any handling of the baby must be of the gentlest, and vigorous attempts at artificial respiration must be avoided ; having made sure that the airway is clear of mucus, traction of the tongue forwards and gentle rhythmic compression of the lower half of the chest must suffice. Inhalations of oxygen and carbon dioxide (5 per cent.) are valuable. Considerable benefit may follow the rectal injection of two ounces of 10 per cent. saline, repeated in four hours if necessary, in order to reduce cerebral œdema (Moncrieff). Convulsions should be controlled with chloral hydrate, the dose being increased until the fits are checked. It may be necessary to give as much as a grain every two hours or even hourly for a few doses.

When the fontanelle is bulging and tense, the cerebro-spinal fluid is blood-stained, and the signs indicate that the hæmorrhage is on the vertex of the brain, surgical intervention should be seriously considered, for success has attended the opening of the skull and the removal of the blood-clot.

It has been suggested that the size of the hæmorrhage may be increased because of the delayed clotting time in the newborn, which is due to the low level of prothrombin in the blood. By giving vitamin K to the mother some four hours before birth takes place, the prothrombin level in her offspring can be considerably raised,[1] and therefore, with the object of diminishing the risk of intracranial hæmorrhage, it has been advised that this vitamin should be given to women in labour, either as a routine, or at any rate to those in whom a difficult labour seems likely.

Spinal Injury

Injury to the spine with hæmorrhage into and around the cord may be produced by severe traction during a breech delivery. The symptoms vary according to the site and extent of the injury; as a rule there is a flaccid paralysis, with abolition of reflexes and loss of sensation, but if the lesion is high in the thoracic region the lower limbs may be spastic.

Fortunately this type of injury is uncommon, for it is always severe, and is usually fatal.

Facial Paralysis

Paralysis of the face may be produced during forceps delivery through injury to the facial nerve as it crosses the ramus of the mandible. As a rule only one side is involved, and the paralysis has the characters of a lower motor neurone lesion. The loss of movement is readily seen when the infant cries, and in repose the eye on the affected side remains half-open. Sucking is not interfered with, because the tongue muscles, which are those chiefly concerned in the act, are supplied by the twelfth cranial nerve.

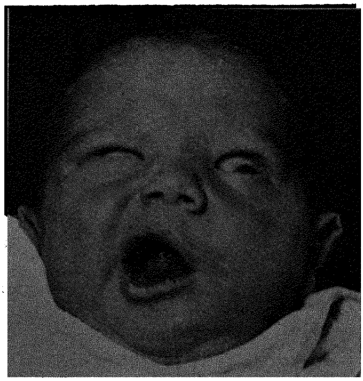

FIG. 2. Left-sided facial paralysis following forceps delivery.

In the majority of cases the paralysis clears up entirely in two or three weeks. It is quite exceptional for it to remain permanently. Treatment is hardly called for, except to protect the conjunctiva until the orbicularis oculi muscle has recovered. If the paralysis continues for more than three weeks, gentle electrical stimulation may be employed.

Facial paralysis due to intracranial damage is rare, but the writer has under his care a boy with paralysis of one side of the face and of the opposite arm and leg, dating from a difficult birth.

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[1] Hellman, L. M., Skettles, L. B., Eastman, N. J., *Amer. Jour. Obst. Gynec.*, 1940, **30**, 844.

Brachial Palsy

Stretching, bruising, and even tearing of the brachial plexus may occur during labour from excessive traction with a wide separation of the head from the shoulder. Generally the fibres springing from the fifth and sixth cervical roots are involved, giving rise to paralysis of the upper-arm type or Erb's paralysis. Much less commonly the fibres from the lower roots—the eighth cervical and first thoracic—are affected, causing paralysis of the lower-arm type or Klumpke's paralysis. Rarely the whole plexus is torn, and this may be associated with hæmorrhage into the spinal cord; and is usually fatal.

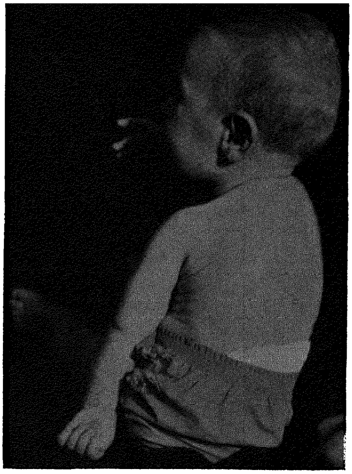
FIG. 3. Erb's paralysis of the left arm.

Erb's Paralysis. As a rule only one arm is affected, and the paralysis is noticed a few days after birth. The muscles deriving their nerve supply from the upper roots of the plexus include the deltoid, spinati, biceps, brachialis anticus and brachioradialis, and when these are paralysed the arm adopts a characteristic position, hanging limply from the shoulder and being internally rotated, with the elbow extended, the forearm pronated, and the palm of the hand directed backwards and outwards.

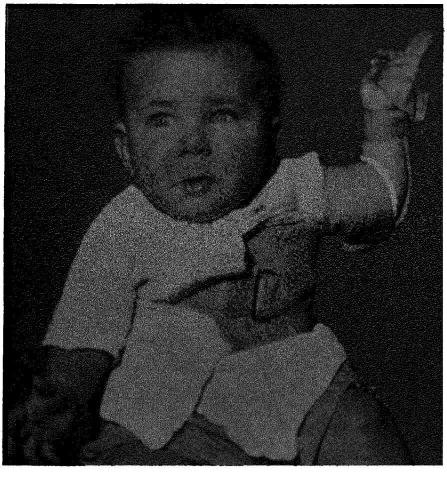
FIG. 4. Erb's paralysis of the left arm, showing the correct method of applying the splint.

Fortunately the majority of cases recover after a period of a few months up to two years. In the rare instances where the roots have been completely severed and the paralysis is permanent, the muscles rapidly waste, and the condition may be confused with acute poliomyelitis, but the history of paralysis from birth should prevent error.

Treatment. As soon as the condition is recognised, a light celluloid splint should be applied in a position which will rest and relax the paralysed muscles. To do this the arm should be abducted to a right angle with the trunk, the shoulder be externally rotated, the elbow flexed, and the forearm supinated.

The splint should remain in use for several months until the child can raise the arm voluntarily. Massage may begin after two or three weeks. If no signs of recovery have appeared after a few months, an attempt at nerve-suture may be made.

Klumpke's Paralysis. The paralysis affects the muscles of the forearm and small muscles of the hand. A light splint should be applied to keep the forearm pronated and the fingers extended.

Sterno-mastoid Tumour

This consists of a hard painless lump in one or other sterno-mastoid, usually towards the lower half of the muscle. The tumour is noticed a few days after birth, and may attain the size of a walnut. It most commonly occurs after breech delivery, and is generally attributed to a hæmatoma within the muscle sheath caused by excessive tension on the muscle with tearing of some of the fibres. Middleton [1] has, however, pointed out that the tumour does not show the ordinary characters of a hæmatoma, being from the first hard and non-fluctuating, and he attributes it to an obstruction of the veins draining the muscle, possibly with thrombosis as well.

The head is often held inclined towards the side of the tumour, but full movement of the head and neck can be passively carried out, and the torticollis is only temporary. A more permanent wryneck may appear after some months or even years, due to subsequent fibrosis of the muscle.

Treatment is hardly necessary beyond daily passive movement of the head and neck to preserve the normal range of movement. The tumour gradually absorbs in a few months.

NEONATAL INFECTIONS

Neonatal Sepsis

The newborn child has practically no immunity against the common pyogenic organisms, and is therefore an easy prey to infection. The most common portal of entry for organisms is the raw wound at the umbilicus, but any skin abrasion is readily infected, and those in charge of an infant may easily convey infection either with their breath by droplets of sputum or on their hands. Owing to the lack of immunity, infection which begins locally may quickly develop into septicæmia or pyæmia, and, because the fighting forces on which the infant can draw are so feeble, the clinical picture is a very different one from that seen in adults. The local lesion may show practically no reaction, nor may there be any striking general effect, and yet the infection may already be widely disseminated. The lack of any well-defined symptoms or signs accounts for many neonatal deaths being either attributed to other causes or passing unexplained ; yet the fact remains that simple infections account for a large number of deaths in early infancy, and the realisation of this must make us all the more insistent upon a proper hygienic regard towards the newborn.

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[1] Middleton, D. S., *Brit. Jour. Surg.*, 1930, **18**, 188.

A mild degree of infection at the umbilicus may give rise to some local redness and swelling, which may clear up with antiseptic dressings, or may go on to the formation of a red pulpy mass of granulations. In more severe cases an abscess may form, and in feeble or premature infants the inflammation may progress to gangrene, which is almost invariably fatal. A general spread of infection from the umbilicus takes place *viâ* the umbilical vein, which may become the site of a septic thrombus. The infection first reaches the liver, where it is likely to cause jaundice, and may then give rise to multiple pyæmic abscesses in various parts of the body, particularly in the lungs and kidneys, or the infant may become septicæmic. There may be an attempt to localise the infection in other parts, giving a variety of septic conditions such as meningitis, pericarditis, broncho-pneumonia, peritonitis, osteomyelitis, or multiple septic arthritis. Abscesses may form in the subcutaneous tissues, especially over points of pressure such as the scalp, scapulæ, buttocks, and heels, and these may extend to involve large areas, or the tissues in these situations may become gangrenous. Gangrene of the extremities—fingers, toes, and genitalia—may also be a complication.

Erysipelas is sometimes met with as a result of infection of the umbilicus or of any superficial abrasion, especially in the region of the external genitalia. The typical raised angry-red rash quickly spreads, and at this age the illness is usually fatal.

The general effect of sepsis is soon evident. Weight is rapidly lost, and the infant becomes wizened and pale. The normal healthy sleep is broken and restless, there is considerable difficulty in getting the infant to take feeds, crying dwindles to a feeble whine, and gradually the baby sinks into a state of semi-coma. The temperature varies; at first it may be raised and the fever may be continuous or swinging, but as the infant grows more feeble the temperature falls and for some days before death is subnormal. The tongue becomes dry, and the respirations are so faint that even if broncho-pneumonia is present its detection is very difficult. The stools become frequent, green, and watery, the fontanelle is depressed and lacks resilience, the skin loses its elasticity, jaundice is common, and there is a greatly increased tendency to bleed either into the skin or from mucous surfaces, giving rise to hæmatemesis, melæna, or hæmaturia. The urine is albuminous, and there may be long periods of suppression. Convulsions usually make a late appearance and are a warning of death ; other infants sink into a moribund state of apathy, lying comatose with eyelids half-open, the respirations become irregular, and at the end there are recurring periods of apnœa separated by a few gasping breaths.

Prognosis. So long as the infection remains localised there is a fair prospect of recovery, but once septicæmia or pyæmia has occurred the outlook is practically hopeless. The advent of jaundice, purpura, multiple subcutaneous abscesses or gangrene points to a fatal result. Some infants linger on for weeks, becoming profoundly emaciated and seeming unable to digest the most simple foods, and eventually die in a condition of marasmus.

Treatment. In view of the gravity of sepsis in the newborn, its prevention must be regarded as vitally important. The principles of asepsis must apply to the infant at birth as well as to the mother, diligent care must be paid to any superficial skin abrasion as well as to the umbilicus, and those in charge of the infant must be free of respiratory infections or of septic skin lesions.

Local sepsis should be dealt with by careful cleansing, and dusting with a sulphanilamide powder. If suppuration occurs, fomentations may be required, followed by free drainage of the pus. When infection has become generalised treatment is less satisfactory. The body heat must be carefully conserved, fluid should be given freely, and every effort must be made to secure a supply of breast milk. Small blood transfusions are sometimes of great value, and they may be repeated, but before deciding to give a transfusion a red cell count should be done, for although the infant may appear dehydrated, the blood may actually be concentrated, and in that case infusions of saline either intravenously or subcutaneously would be of greater benefit. Stimulant treatment by weak mustard baths (half an ounce of mustard to one gallon of water) may have a rallying effect, and injections of stimulants such as camphor or strychnine may be required. If multiple small subcutaneous abscesses develop they should be freely opened, and the infant should be held twice a day in a warm antiseptic bath, such as a 1 in 4,000 potassium permanganate or a weak iodine bath. Erysipelas should be treated by painting the skin in front of the rash with 10 per cent. ichthyol, or a compress of ichthyol ointment may be applied, and one of the sulphanilamide group of drugs be given by mouth, in doses according to the table on p. 715.

Pemphigus Neonatorum (Bullous Impetigo of Infants)

Pemphigus neonatorum consists of a widespread bullous eruption of the skin usually due to staphyloccal infection, and may be looked upon as the infantile form of impetigo.

The rash appears generally towards the end of the first week, beginning as small blebs on any part of the skin surface. At first the blisters are small, but quickly increase in size, and several may coalesce to cover areas of 2 or 3 inches across. They are filled with a clear serous fluid, and soon rupture, leaving a raw bright red area with loose shreds of epithelium attached at the edge. Less often the bullæ dry up into scabs, or may suppurate.

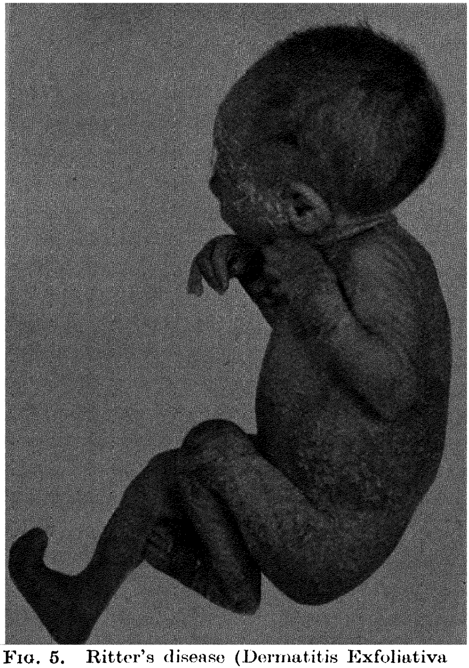

FIG. 5. Ritter's disease (Dermatitis Exfoliativa Neonatorum) in an infant aged two weeks.

The condition is usually met with in

feeble infants, and signs of grave constitutional disturbance are generally evident. There may be high fever, but in the most severe cases there may be scarcely any reaction, and the temperature is then subnormal.

Diagnosis. This is seldom difficult. A bullous eruption may occur in congenital syphilis, but the rash tends to be confined to the hands and feet, and there are likely to be other signs such as snuffles, condylomata, or enlargement of the liver and spleen. The condition described as Ritter's disease, or Dermatitis Exfoliativa Neonatorum, is now recognised as being essentially the same as pemphigus neonatorum, but clinically bleb formation is less in evidence, while shredding and exfoliation of the skin are more extensive.

Prognosis. In 297 cases collected by Ritter the mortality was 48 per cent. Death occurs from exhaustion or from septicæmia.

Treatment. Neonatal pemphigus is highly contagious, and therefore the infant must be isolated. This applies with particular force in lying-in institutions.

Treatment is conducted on antiseptic lines. Each bulla should be broken and the loose epidermis cut away. The raw surface can then be dusted with an antiseptic powder such as equal parts of calomel and boracic powder, or dressed with 2 per cent. ammoniated mercury ointment. A useful plan is to have the baby held in a warm antiseptic bath three or four times a day, and for this purpose nothing is better than a 1 in 4,000 bath of potassium permanganate (pale purple in colour). Favourable reports have followed sulphanilamide therapy. The general measures which should be taken have been described under Sepsis Neonatorum.

Ophthalmia Neonatorum

This was at one time a prevalent cause of blindness, but where prophylactic treatment has been employed at birth the condition has become rare. The most common infecting agent is the staphylococcus, but other cocci, diphtheroids, and Koch-Weeks bacilli may be responsible ; the gonococcus is the most important organism, and accounts for roughly a third of the cases. Infection of the eyes takes place during labour by direct contact with infected maternal discharges. In institutions the infection may be easily passed from one infant to others by those in attendance, and therefore as soon as an infant is seen to be infected strict isolation must be enforced.

Symptoms. To begin with only one eye may be affected, but usually both are involved. On the second or third day after birth the eyelids become swollen and red, and may be stuck together by exudate. The discharge quickly becomes turbid, yellow and abundant, and from it the infecting organism can be isolated. Unless prompt treatment is instituted, there is a considerable risk of the infection spreading to involve the cornea, and this may result in a varying degree of blindness. More severe cases may go on to panophthalmitis with disorganisation of the eyeball. Under effective treatment the discharge rapidly lessens in amount and becomes thinner, and ceases in three or four weeks. In gonococcal cases arthritis may be a late sequel.

Prevention. The method introduced by Crédé is now widely used. This consists of instilling one drop of 1 per cent. silver nitrate into each eye immediately after birth. A drop of 10 per cent. protargol may be used instead, with as good results. Occasionally the drops give rise to sufficient irritation of the conjunctiva to produce a slight discharge for a day or two.

Treatment.[1] Prompt isolation is essential. Those handling the infant must observe strict asepsis, and should wear rubber gloves. If only one eye is involved, the infant should lie with the unaffected eye uppermost to prevent infected discharge trickling into it, and the healthy eye should be bathed daily and a drop of 5 per cent. protargol be instilled.

The efficacy of sulphapyridine has replaced the older methods. The dose should be according to the table on p. 715, and should be continued until twenty-four hours after a clinical cure has been obtained, which usually means treatment for three to five days. Local treatment should consist of three-hourly irrigations with 0·5 per cent. saline solution followed by a drop of medicinal paraffin to prevent the eyelids from sticking together. If the cornea becomes steamy, a drop of 1 per cent. atropine sulphate should be instilled three times daily.

Tetanus Neonatorum

In this country tetanus in the newborn is fortunately a rare condition, for it has a high mortality, but it is still to some extent endemic in other parts of the world. Infection with the tetanus bacillus takes place *viâ* the umbilicus, and is sometimes due to the ritual treatment of the umbilical stump with dirt.

Symptoms. The onset is generally towards the end of the first week, but may be delayed until a fortnight after birth. The first symptom is difficulty in sucking owing to trismus of the jaws. This is followed by a general rigidity of the trunk and limbs, and within a day or so typical tetanic spasms occur. In these the infant passes into such severe rigidity that it is possible to raise him horizontally from the knees, the arms and legs are extended, and the fingers are so tightly clenched that the hands cannot be opened, the face assumes a " risus sardonicus " in which the eyes close to mere slits and the corners of the mouth are drawn outwards and downwards (there is little sardonic about this), the respiratory muscles become fixed and there is increasing cyanosis. After a minute the spasm passes off, and the limbs may then twitch a little. Rigidity does not, however, pass off completely between the spasms, and the abdominal wall in particular remains taut. In severe cases spasms recur frequently and are brought on by any movement, so that feeding and nursing become very difficult. The temperature is raised, and may be hyperpyrexial.

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[1] Sorsby, A., *et al.*, *Brit. Med. Jour.*, 1942, *ii.*, 323.

The diagnosis does not present difficulty if the possibility of the condition is borne in mind. Spasmodic contraction of muscles may also occur in intracranial injury, but even so the condition does not resemble tetanus. Cameron has, however, pointed out how closely some cases of septic meningitis in the newborn may simulate tetanus, especially when the cervical spinal meninges are chiefly affected.

Prognosis. The mortality is very high, in the region of 90 per cent. Hope of recovery can hardly be entertained unless the onset of symptoms has been delayed until the end of the second week, and in the fatal cases the disease usually runs a short course of two or three days. The first indication of improvement under treatment is a lengthening interval between the paroxysms.

Treatment. The infant must be disturbed as little as possible. Feeds are best given by a catheter passed through the nose. The umbilicus should be cleansed and treated with antiseptics. After a preliminary skin test to exclude sensitivity to horse serum, 10,000 to 20,000 units of anti-tetanic serum should be given, half the dose being injected intravenously and the remainder intramuscularly. Sedatives in relatively large doses should also be given ; for this purpose rectal injections of paraldehyde, allowing 4 minims per lb. of body weight, are most useful ; or chloral may be tried by mouth, giving two grains every two hours for six doses, and continuing according to the condition. If the drug is not retained, double the dose may be given by the rectum.

Subcutaneous injections of magnesium suphate (5 c.c. of a

4 per cent. solution injected every four hours) have been recommended to curtail the paroxysms.

TETANY IN THE NEWBORN

Of recent years several cases of tetany in newborn infants have been described, associated with a low calcium level in the serum. While in some instances there have been typical carpo-pedal spasms, and Chvostek's sign of facial irritability and Trousseau's sign (see p. 132) have been demonstrable, in others convulsions have been the outstanding feature, with or without laryngeal spasm.

An infant with this condition under the author's care [1] began attacks of twitching, culminating in convulsions, on the tenth day after birth. The serum calcium was as low as 5·8 mgm. per 100 c.c. Treatment by injections of calcium gluconate combined with vitamin D by mouth was only partly successful ; the most effective therapy consisted of five daily injections of parathormone, a total of 14 units being given.

ICTERUS NEONATORUM

Jaundice occurs in the newborn under the following conditions :—

Simple or physiological jaundice.
Familial icterus gravis.
Infective jaundice.
Syphilitic jaundice (see p. 651).
Familial acholuric jaundice (see p. 443).
In association with congenital obliteration of the bile ducts (see p. 254).

The first three varieties will be considered in this chapter.

Simple or Physiological Jaundice

Jaundice of a mild degree, but discernible to the naked eye, is of common occurrence in the newborn. It appears on about the second day and begins to fade at the end of a week, and has generally disappeared by the time the infant is a fortnight old. The stools do not become pale, indicating that the jaundice is not obstructive, and the urine does not contain bile pigment, indicating that the degree of jaundice is but slight. The health of the infant is unaffected, and the jaundice leaves no ill effects. As to the incidence of this type of jaundice, it has been estimated that from half to three-quarters of all newborn infants are affected, and it is particularly noticeable in premature or small babies. Sander[1] found among 1,500 newborn infants that the lower the birth weight, the greater was the degree of jaundice, and Kramer[2] has shown that the amount of bilirubin in the blood of the newborn is invariably increased, that is to say, in all infants the blood is icteric, but the bilirubin only rises high enough to produce obvious jaundice in from half to three-quarters of them. The Van den Bergh test gives an indirect positive reaction.

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[1] Lloyd, O., *Arch. Dis. Child.*, 1938, **13**, 275.

Various explanations have been put forward to account for the jaundice. It has been attributed to an unusual degree of viscidity of the bile, although the evidence points to the icterus being non-obstructive. It is usually accounted for by the liberation of bilirubin consequent upon the extensive destruction of red blood cells which takes place immediately after birth. The blood of the newborn shows a red cell count in the neighbourhood of 6,500,000 cells per c.mm., but at the end of the first week the figure has fallen to about 4,500,000. There is evidence that the cells that are destroyed are mostly immature nucleated forms, which have been pressed into the fœtal circulation in order to prevent anoxæmia of the fœtal tissues, since the circulation at that time consists of a mixture of arterial and venous blood. The changes that take place in the circulation at birth ensure that the tissues shall be supplied with pure oxygenated blood, and the need for these extra cells no longer exists.

No treatment is required.

Familial Icterus Gravis

This is a condition which often affects successive infants in a family, although isolated instances also arise. Males and females are equally affected. Rolleston was able to collect 120 instances in 25 families. It not uncommonly happens that the first child escapes, while later members are all affected.

The jaundice appears earlier than simple physiological icterus, and may even be present at birth. It rapidly deepens, and the infant quickly becomes drowsy, the liver and spleen become enlarged, purpuric hæmorrhages may appear in the skin and mucous membranes, convulsions may occur, and death takes place after a few days. As a rule the stools are bile coloured throughout the illness, but occasionally they become pale for the first few days, probably owing to a temporary biliary obstruction from the increased viscidity of the bile, which in turn is due to the excessive hæmolysis that is taking place. Bile pigments appear in the urine. The Van den Bergh test is variable. The number of nucleated red cells in the circulation is considerably raised, and may reach as high as 25,000 per c.mm. ; use may be made of this if the diagnosis is in doubt, for the appearance of numerous erythroblasts on microscopical examination of a drop of blood can be taken as confirmatory evidence. If recovery takes place a severe anæmia follows, and may persist for several months. A relapse of jaundice after apparent recovery has been recorded.[1] In not a few cases recovery has been followed by mental defect, spastic paralysis, and fits. The development of severe skeletal deformities, due to osteitis fibrosa with multiple fractures, has been reported as a sequel, but is fortunately rare.

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[1] Sander, *Ztschr. f. Kinderh.*, 1927, **43**, 434.

[2] Kramer, P. H., *Nederl. Tijd. v. Geneesk.*, 1926, **1**, 249.

Post-mortem examination shows little to the naked eye beyond severe jaundice of the viscera and perhaps purpuric hæmorrhages. For the most part the nervous system does not show bile-staining, but the region of the basal ganglia is sometimes jaundiced, a condition to which the name Kernicterus has been applied. The selective staining of the nervous system probably accounts for the occasional nervous sequelæ.

Histologically the most typical feature is the presence of numerous areas of extra-medullary hæmopoiesis. This is particularly noticeable in the liver and spleen, but has also been described in many other situations, and the marrow is also hyperactive. The centres of blood formation show numerous nucleated red cells. To this histological picture the name Erythroblastosis Fœtalis has been given, and it serves to link together three clinical conditions, namely, hydrops fœtalis (characterised by universal œdema in a fœtus which is generally still-born), icterus gravis neonatorum, and hæmolytic anæmia of the newborn, in each of which it is present.

Recent work [2] indicates that both icterus gravis and congenital hæmolytic anæmia arise from the presence of an agglutinin in the mother's serum incompatible with the infant's red cells, which consequently undergo hæmolysis far in excess of that occurring in the normal infant. The state of erythroblastosis fœtalis is called forth in response to this hæmolysis.

Treatment. The introduction of blood transfusion has superseded all other methods, and has reduced the mortality in a remarkable way. Roughly 15 c.c. of blood should be given for each pound of the infant's weight, the transfusion being repeated if necessary every fourth or fifth day until the red cell count has returned to normal. Although the mother's blood is often used with eventual success, more satisfactory and lasting results may follow blood obtained from a donor outside the family, but the recent investigations, to which reference has been made above, indicate that this will only be so provided the donor's blood does not contain the Rhesus factor, *i.e.*, is Rh negative, and a donor who is Rh negative should therefore be selected. Every effort should be made to secure a diet of breast milk. For several weeks after recovery, a small dose of iron, such as Iron and Ammonium Citrate, gr. 3, daily, should be given.

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[1] Hawksley, J. C., and Lightwood, R., *Quart. Jour. Med.*, 1934, 3, 159.

[2] Boorman, K. E., Dodd, B. E., and Mollison, P. L., *Brit. Med. Jour.*, 1942, *ii.*, 535.

Infective Jaundice

Infective jaundice in the newborn arises most commonly from the umbilicus, infection passing along the umbilical vein and sometimes giving rise to multiple small pyæmic abscesses throughout the liver. Occasionally a more diffuse hepatitis occurs.

In addition to jaundice, which may not come on until a week or ten days after birth, there are likely to be the symptoms of neonatal septicæmia or pyæmia, which have already been described. The outlook when jaundice has developed is always grave. Treatment must be directed primarily to the septic condition, but in view of the evident damage to the liver a plentiful supply of fluid and sugar is desirable, and if vomiting or drowsiness prevents the infant taking properly by mouth, glucose salines should be given subcutaneously or intravenously.

Two special forms of infective jaundice in the newborn have been described, namely, Buhl's disease and Winckel's disease. The former is characterised by progressive jaundice, hæmorrhages, œdema, and death, and at autopsy there is widespread fatty degeneration. The latter may arise in epidemic form in lying-in institutions, although it has not occurred for many years in this country. In addition to the symptoms outlined above, hæmoglobinuria is a usual feature. Both conditions are really examples of severe sepsis in the newborn, and are no longer regarded as specific diseases.

ANÆMIA IN THE NEWBORN

The most common form of severe anæmia in the newborn is found in association with erythroblastosis fœtalis, and termed congenital hæmolytic anæmia. An acute aplastic type of anæmia is occasionally met with, in which the red blood cells and hæmoglobin fall rapidly, while evidence of regeneration, such as an increase of nucleated red cells and reticulocytes in the circulation, is lacking. Although these cases are usually fatal in the first few weeks, repeated blood transfusions should be given in the hope that the marrow may eventually recover its function. Acholuric jaundice must also be borne in mind as a rare cause of severe anæmia developing shortly after birth. Anæmia may be the result of traumatic hæmorrhage, and occurs under conditions of spontaneous bleeding, usually from the gastro-intestinal tract or umbilicus, which are more fully described under "hæmorrhagic disease of the newborn."

Congenital Hæmolytic Anæmia

This form of anæmia is due to excessive hæmolysis taking place at birth and for a few weeks afterwards. Reference to it has already been made in connection with icterus gravis neonatorum, where it was pointed out that recovery from the jaundice is often followed by severe anæmia. In other cases there is no history of jaundice, and the child is first seen when about a fortnight old on account of anæmia. The position is that jaundice occurs when the hæmolysis is so rapid that the infant is unable to excrete the excess of bilirubin quickly enough, while when the hæmolysis is less acute but more persistent, anæmia is the first symptom, but the underlying process is essentially the same.

Examination of the blood shows a hyperchromic anæmia, the red cells being reduced to a relatively lower level than the hæmoglobin. They may fall below 1,000,000 per c.mm. That the blood-forming centres are attempting to combat the anæmia is indicated by a considerable reticulocytosis and by the presence of numerous nucleated red cells in the circulation. The spleen may be considerably enlarged, and the urine contains an excess of urobilinogen and urobilin.

In fatal cases the bone marrow has the appearance of having been the scene of great activity, and extra-medullary centres of blood formation are found in the liver and spleen (erythroblastosis fœtalis).

Treatment. Recovery depends on making the diagnosis of the type of anæmia early, and then giving whole blood either intravenously or intramuscularly. Apart from a history of jaundice, the characters of the blood picture—a hyperchromic anæmia with reticulocytosis and an erythroblastæmia—are sufficient to establish the diagnosis. The giving of blood[1] is urgent.

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[1] In addition to compatibility, the donor should be Rh negative.

It is best given as a transfusion, which should be repeated every fourth or fifth day until the blood picture approaches the normal. If the technical difficulties of transfusion in so young a subject cannot be overcome, a daily injection of 10 c.c. of whole blood should be given deeply into the muscles, and should be continued until the blood picture shows a progressive improvement, but it must be understood that blood by this route is much less effective than when given by transfusion.

Hæmorrhagic Disease of the Newborn

In newborn infants the prothrombin level in the blood falls during the first four or five days after birth, and then gradually returns to normal. If the prothrombin level falls as low as 15 to 20 per cent. of normal, spontaneous hæmorrhages are likely to occur, and their clinical manifestations are embraced in the term "hæmorrhagic disease of the newborn." A less severe lowering of the prothrombin, while not leading to spontaneous hæmorrhage, will tend to prolong bleeding arising from other causes, such as trauma received during birth, or hæmorrhages associated with the various septic disorders of the newborn.

In 1936, the discovery by Dam and his co-workers in Copenhagen, of a fat-soluble substance, vitamin K,[1] which would rapidly restore the prothrombin level to normal, has given a means of controlling the various forms of hæmorrhagic disease of the newborn. The most important situation for such hæmorrhage is from the gastro-intestinal tract (hæmatemesis and melæna neonatorum); others that will be mentioned are the vagina, urinary tract, and umbilicus.

Vaginal Hæmorrhage. Bleeding may take place from the vagina, without hæmorrhages from other parts. The bleeding usually starts within twenty-four hours of birth and may continue for two or three days, but it is, as a rule, mild in extent, and treatment is not required.

Hæmaturia. The loss of blood is usually slight. An instance was that of a male infant who, when a day old, was noticed to have stained the napkin pink. This was at first thought to be due to urates, but during the next three days frequent small specimens of almost pure blood were voided per urethram. The bleeding then ceased, leaving the infant little the worse.

Mention has already been made of fatal hæmorrhage into the lungs—acute hæmorrhagic pneumonia (p. 12).

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[1] Vitamin K is a naphthoquinone derivative. Synthetic analogues are even more potent than the original vitamin, and the preparation now in common use as vitamin K is 2-methyl-1 · 4 naphthoquinone.

Umbilical Hæmorrhage. Septic conditions of the umbilicus may of course give rise to hæmorrhage, but even without any signs of sepsis severe and even fatal bleeding may also occur. The loss of blood begins at about the end of the first week, and consists of a steady oozing, which in a day or two may exsanguinate the infant, for it must be pointed out that at this age the loss of an ounce or two of blood amounts to a very considerable hæmorrhage. The condition should be treated by applying firmly a wad of gauze soaked in adrenalin, and at the same time 5 mgm. of vitamin K (see opposite) should be given by mouth. If the loss of blood has been severe, transfusion of blood will be necessary.

Hæmatemesis and Melæna Neonatorum. The gastro-intestinal tract is the most common source of spontaneous hæmorrhage in the newborn. Both sexes are equally liable, and the incidence has been estimated as 1 in 300 to 600 births. The infants have, as a rule, been born at full term, and until the typical symptoms appear have seemed in perfect health.

Symptoms. The onset is sudden. The first symptom in most cases is the passage of a dark tarry stool on the first or second day after birth. The stools that follow contain more blood, and may in fact consist of practically pure blood. Hæmatemesis occurs in about half the cases, but usually does not precede the melæna. The temperature may be raised, but is as often subnormal, the pulse-rate rapidly mounts and may become uncountable, the infant soon appears pale and collapsed, and unless treatment is quickly given death will probably occur in from a few hours up to two or three days after the first symptom, indeed it has been known to occur before there has been time for blood to appear in the stool or the vomit, post-mortem examination showing a large amount of blood in the stomach and intestine.

Theories of Causation. Careful examination at autopsy shows, in something less than half the fatal cases, one or more minute ulcerations in the gastric or duodenal mucosa, but this is a not sufficiently common finding to be conclusive. Even if it is the most probable cause, the origin of the ulceration is not at all clear. Asphyxia during birth, leading to circulatory stagnation and thrombosis, has been suggested, as has a mild gastro-intestinal infection. Once bleeding has commenced, a low prothrombin level will allow it to continue unabated.

Treatment. This consists of giving vitamin K, combined with injections of whole blood. The benefit of the latter is well proven, and is due in part to direct improvement of the anæmia, and in part because blood offers a rapid method of supplying prothrombin. Blood injections to be effective must be given promptly. For mild cases, the intramuscular injection of 10 c.c. of blood into each buttock, followed by half this amount if bleeding still continues, is often sufficient, but in the more severe cases intravenous transfusion is required. This procedure calls for more technical skill than intramuscular injection, and the compatibility of the donor's and recipient's bloods must first be established, but there is no doubt that blood given intravenously exerts a much quicker influence on the prothrombin level.

Vitamin K improves the prothrombin level more slowly than does blood transfusion, but its effect is more lasting. The vitamin should therefore be given at the outset of treatment. It is active by mouth, and 5 mgm. (contained in 7 minims of Kapilon, or in half a tablet of Synkavit) should be given twice a day for two or three days.

The infant must be kept as quiet as possible and should not be picked up for any reason, nor be put to the breast for twenty-four hours after the last appearance of blood. There is, however, no advantage in withholding fluids, and one ounce feeds of albumen water at two-hourly intervals will help to allay restlessness. Drugs by mouth, other than vitamin K, are of doubtful value, but two drops of adrenalin in a teaspoonful of water may be given hourly for five or six doses.

The loss of blood will inevitably lead to anæmia, which should be dealt with during convalescence by giving iron and ammonium citrate (gr. 2 t.d.s.). When recovery takes place it is complete, and there is no particular tendency to hæmorrhage later in life.

Œdema in the Newborn

Several varieties of œdema may be met with in the newborn.

Hydrops Fœtalis. Severe generalised œdema, together with large collections of fluid in the serous cavities, may develop during fœtal life, and leads either to still-birth or to death within a few days of birth. More than one pregnancy in a family history may terminate in this way. The condition has recently received fresh interest because of its association with familial icterus gravis and congenital hæmolytic anæmia, for these three diseases share in common a high erythroblast count in the circulation, and at autopsy show numerous extramedullary centres of active blood formation.

Œdema may be due to compression of the superficial vessels during labour. The caput succedaneum is the most common example of this, but considerable œdema of the perineum and external genitalia may also appear at birth, and has been ascribed to the same cause. The œdema is transient, lasting only a few days, but occasionally the swollen tissues become infected, which for obvious reasons is more likely to happen in the perineal region, and the inflammation may go on to local gangrene.

A pitting œdema beginning in the eyelids or at the extremities, and thence spreading until it becomes generalised, may develop in premature infants or in those of a weak and puny constitution. It is probable that cold is a direct factor in causing the œdema, at all events it is not likely to disappear until the temperature has been maintained at 98° to 99° F. for a day or two, and there is likely to be other evidence of a low vitality such as shallow respirations and a feeble cry. It has recently been shown [1] that the excretion of salt through the kidney in newborn infants, and particularly in those prematurely born, is restricted, rendering them more than ordinarily prone to œdema, and incidentally calling for moderation in the giving of saline. These infants must be managed on the same lines as a premature baby, particular care being taken to preserve the body heat.

Familial Œdema (*Milroy's Disease*). This is a variety of œdema which is confined to the legs. It is painless, pits on pressure, and is persistent. Successive members of a family may show the condition, and there may be a history of it in previous generations.

Treatment is not generally required during childhood. The œdema can be reduced by firm bandaging before the child gets up, and by massage, but the swelling returns when treatment is stopped.

Congenital Elephantiasis. A non-pitting œdema of one or more limbs may be due to lymphangiectasis, and may be associated with a diffuse nævoid condition of the skin. This is more fully considered on p. 461.

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[1] Young, W. F., Hallum, J. L., McCance, R. A., *Arch. Dis. Child.*, 1941, **16**, 243, and 1942, **17**, 65

CHAPTER III

THE PREMATURE INFANT

THE infant mortality rate (deaths in the first year) has fallen during this century by more than half, but the neonatal mortality rate (deaths in the first month) has scarcely altered, and in this country is still about twenty-eight deaths per 1,000 live births. That the neonatal death rate can be lowered has been clearly shown, for instance in Adelaide, South Australia, in 1936, it was only twenty, and in 1937 in Amsterdam (Holland) and Göteburg (Sweden), it was as low as sixteen per 1,000 live births. At the present day roughly half the deaths occurring in the first year take place during the first month, and of these a little more than half affect prematurely born infants ; a considerable proportion of these deaths are due to preventable conditions such as infections, chilling, failure to obtain breast milk, and digestive difficulties arising from artificial feeding.

Although a fœtus born after the 28th week of pregnancy is considered to be viable, the chances of survival are poor before the eighth month of pregnancy. Some of those who are born between the end of the eighth month and full-term are sufficiently well developed not to require special treatment, and can be managed in the same way as an infant born at full-term, while on the other hand some infants born at term are so puny and weak as to need all the special care that is given to premature infants. In general terms the weight at birth is a more reliable index of prematurity than the supposed length of gestation ; when the birth weight is below 5½ lbs. the infant should be given the particular care that premature birth demands.

The cause of premature birth is in most cases unknown. This was so in 60 per cent. of 437 premature infants studied by Capper. Twin pregnancies are more likely to terminate prematurely than are single pregnancies, and account for between 15 and 25 per cent. of premature births. Congenital syphilis is occasionally the cause, but is more likely to give rise to a still-birth than to the premature birth of a living child. Extensive analyses by Capper and Ylppo showed syphilis as the cause in about 3 per cent of cases. Illnesses in the mother, such as eclampsia, nephritis, heart disease, the acute infectious fevers, lead poisoning, etc., may also account for a premature labour.

The chance of survival is directly proportional to the age of the infant—or rather fœtus—at the time of birth, and to the birth weight, but of these two factors the first is the more important; that is to say, a small infant born near term is more likely to survive than a larger child born more prematurely. The following tables show how the prognosis varies with these two factors.

The likelihood of survival is also affected by the health of the mother during her pregnancy. When for instance a premature birth is due to eclampsia, syphilis, or one of the infectious fevers, the infant is handicapped to a greater extent than if the prematurity is due merely to the presence of twins. When illegitimacy is coupled with prematurity, the infant mortality is almost doubled.

Table showing the chance of survival according to the length of the pregnancy[1]

Length of Pregnancy	Percentage of Infants saved (without incubators)
6 months	0·0
6½ ,,	29·5
7 ,,	39·0
7½ ,,	54·0
8 ,,	78·0
8½ ,,	88·0

Table showing how the chance of survival varies with the weight at birth[2]

Birth Weight	Percentage Mortality		
	1st day	1st month	1st year
Under 1,000 gms. (1 lb. 14 oz.)	37·8	83·8	91·9
1,001 to 1,500 gms. . .	15·7	49·4	64·0
1,501 to 2,000 ,, . .	6·8	24·0	41·0
2,001 to 2,500 ,, . .	1·9	15·4	28·8

Prematurity carries with it certain other risks. There is no doubt that the likelihood of injury to the brain and spinal cord by hæmorrhage produced during birth is greater among premature babies, and is attributable to an increased fragility of their blood-vessels.′ Premature infants are also more prone to develop both anæmia and rickets than are full-term infants. During pregnancy the fœtal liver steadily accumulates a reserve of iron, which is drawn upon by the infant so long as he is receiving a purely milk diet. If the storage of iron is interfered with for any reason, such as by premature birth, the liability to anæmia during the early months of life becomes much greater. The amount of blood destruction that normally takes place immediately after birth is also greater in premature infants, and after two or three months the red cells may have fallen as low as three million per c.mm. One result of this anæmia is to render the infant more prone to infections, particularly of the respiratory and intestinal tracts, and special care needs to be taken of premature infants to protect them from these complications. The increased susceptibility of premature infants to rickets is probably accounted for in part by the deficient storage of calcium salts at the time of birth—accumulation of these salts being made principally in the last two months of gestation—and in part by the difficulty of getting the premature infant to take a diet sufficiently nutritious to prevent the development of rickets. Additions of cod-liver oil to the diet, or of concentrates of vitamin D, must be made from an early age.

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[1] Tarnier, quoted by Holt: "Diseases of Infancy." 1922.

[2] Ylppo .*Klinische Wochenschrift*, 1922, 1, 1241.

The premature infant differs in appearance in many respects from the full-term baby. There is practically no subcutaneous fat, and the skin, which is redder than usual, is wrinkled and gives to the face a peculiarly wizened look. The jaundice of the first few days is more noticeable, and is likely to persist longer than in the full-term infant. The nails are often not fully developed. The low vitality of the baby is shown by the feebleness of the cry, the shallowness of the respirations—which may be at times difficult to perceive, and may not be strong enough to expand the lungs fully, so that at autopsy pulmonary atelectasis is a common finding—by the slow and infrequent movements of the limbs, and by the deficient power of sucking. The infant is unusually drowsy, and passes almost the whole day asleep. Attacks of cyanosis with periods of apnœa are common, and may be simply a part of the general feebleness or may be due to intracranial hæmorrhage. The lack of subcutaneous fat gives rise to an increased loss of heat, so that the body temperature is low, and constant nursing attention must be given to maintain the temperature as near as possible to normal.

Management. The management of premature infants calls for considerable medical and nursing skill. It is essential to conserve the body heat, to give sufficient nourishment, and to prevent infections.

Warmth. Attention to warmth should start immediately after birth, the baby being placed at once in a cot previously warmed and made ready. The infant's temperature undergoes quick fluctuations, because the heat-regulating mechanism is imperfectly developed, and hyperthermia, leading to dehydration, may be easily produced unless the temperature is taken regularly. It should be taken hourly at first until a stable level has been reached, and thereafter every four hours. The rectal temperature should be kept not lower than 98° F. Special low-recording thermometers can be obtained which register down to 85° F., and are a great asset.

A question that will often arise is whether the infant should be nursed in a specially constructed incubator. The advantage of an incubator is that the temperature of the atmosphere surrounding the baby can be easily regulated, and so the risk of chilling is absolutely avoided. But, on the other hand, it is difficult to get an adequate change of air, and this is necessary as it affords a valuable stimulus to the respirations. The use of incubators also calls for constant watching and skilled nursing, and is more easily managed in special institutions than in private homes. On the whole, it is preferable not to employ an incubator.

The infant should be nursed in a cradle lined with a blanket, placed near a coal fire, and protected from draughts by a screen. A thermometer should hang on the cradle, and the temperature of the room should be kept between 70° and 75° F. The cradle itself should be warmed by hot-water bottles placed among the blankets, and, in order to avoid any risk of burning the infant, there should always be two thicknesses of blanket between the bottles and the baby. The cradle may also be warmed by hanging electric light bulbs inside, or by the use of a special electrically warmed blanket, which should be arranged above rather than under the infant so as to avoid any risk of burning. By one of these means the temperature under the bedclothes should be kept at about 80° F. In so warm an atmosphere, drying of the air must be guarded against, otherwise the infant may become dehydrated. This can be prevented by keeping an open pan of water in the room.

The clothing of the premature infant is an important matter. The body and limbs should be loosely wrapped in cotton wool, and a woollen jacket, which does up down the front so that it can easily be removed, should be worn. The legs should be covered with a flannel wrap-over, and the head protected with a flannel bonnet, the face being left free. Instead of the ordinary napkin, a square of absorbent wool should be used to receive the discharges. The infant should be disturbed as little as possible, although gentle changes of position every now and then will assist proper ventilation of the lungs. In order to avoid chilling, the daily bath should be omitted, and in its place the baby should be gently oiled all over with warm olive oil. Bathing in water should be deferred until the weight has reached 7 lbs.

Thyroid has proved very useful as a further means of maintaining warmth. The daily dose should be gauged by allowing gr. $\frac{1}{20}$ of Thyroideum Siccum for each lb. of body weight. Its virtue probably lies in its stimulating effect on metabolism.

Feeding. Premature infants who weigh as much as 5 lbs. at birth are generally capable of sucking quite strongly, and they should then be put to the breast in the ordinary way, beginning twelve hours after birth. For the first two or three days until the flow of milk is established, it will be necessary to supplement the feeding, and for this purpose breast milk should be obtained if it is at all possible and be given from a spoon, otherwise one of the milk mixtures mentioned later must be used. If the infant is born in a lying-in institution there should not be the slightest difficulty in obtaining breast milk from another woman. When the infant can only suck feebly, the establishment of lactation is likely to take a day or two longer than usual.

If the baby is so weak that he is unable to suck, he must be fed by dripping the milk slowly on to the back of the tongue. For this purpose a medicine dropper or a fountain-pen filler or a special Breck feeder (which amounts to a pipette with a rubber teat attached) can be used. Sometimes when the infant is very feeble he may be inclined to choke if fed in this way, and then it is much safer to pass a small catheter into the œsophagus and feed slowly through the tube, allowing five minutes for the milk to pass into the stomach. The catheter should not pass through the cardiac orifice, otherwise vomiting may easily occur. There is practically no danger of the tube getting into the trachea, and it can be passed with hardly any disturbance, so that the method is one which can be safely recommended. It should only be necessary to use it for a few days until the infant can swallow properly.

The feeds will have to be smaller, and must be given at more frequent intervals, than in the case of a full-term infant, while the total amount of food will need to be relatively greater. It is pointed out elsewhere that a healthy full-term baby needs up to two and a half ounces of food for each pound of body-weight per day, but a premature infant will need at the very least three ounces per pound. Feeding must be continued through the night, and in the feeblest infants it is often necessary to give hourly feeds. Although hourly feeds may enable sufficient nourishment to be taken, if an equivalent daily intake can be tolerated by two-hourly feeding, the longer interval should be employed, because it entails less interference with sleep.

It will not be possible at first to give as much as three ounces of food for each pound of body-weight, in fact it may take two or three weeks before the infant can tolerate this amount. The danger of overfeeding a premature infant is as real as that of underfeeding, for the digestion is but feebly developed, and if it should be overtaxed the life of the infant is seriously jeopardised.

Special directions are necessary for the feeding during the first two or three weeks. On the first day the infant should be fed three times ; the subsequent feeding will depend on the degree of prematurity. When the birth weight is below 4 lbs., feeding may have to be hourly during the day and two-hourly during the night (eighteen feeds in twenty-four hours). A drachm at each feed may be as much as the infant will tolerate at first. An increase of half a drachm per feed should be made every other day, so that by the tenth day each feed amounts to approximately three drachms. A careful watch must be kept for signs of overfeeding, such as regurgitation of some of the feed, or more definite vomiting, or diarrhœa, or cyanotic attacks after the meals. These would certainly prevent any further addition to the diet ; indeed a slight reduction for a day or two would be advisable.

At about the end of the second week it should be possible to lengthen the intervals between the feeds. Meals should be given every two hours by day and three-hourly during the night (ten feeds in twenty-four hours). The amount will have to be increased, half or even three-quarters of an ounce being given at each feed. This change in the times of feeding should only be made provided that the weight is showing a slow steady rise. By the twenty-first day the amount should be worked up to an ounce per feed, without, however, relaxing the watch against overfeeding.

After this stage has been reached, the need for further increases will be indicated by the infant's weight—and here may be pointed out the great value to be attached to daily weighing of the baby, so that the feeding may be modified according to the infant's progress. When the feeds have been worked up to ten ounces in twenty-four hours, this amount should be persisted with until the weight begins to falter. Then each feed should be increased by a drachm every day until the rise in weight is resumed. By the time the weight has reached 5½ lbs., three-hourly feeding should begin, omitting one feed at night (seven feeds in twenty-four hours).

The directions that have just been given apply to the majority of premature infants with a birth weight below 4 lbs., but each case needs separate study. When the birth weight is below 3 lbs. the diet may have to be developed more gradually. If the weight at birth is between 4 and 5½ lbs. it is usually possible to begin by feeding two-hourly in the day-time and three-hourly at night, starting with half an ounce at each feed and working up to an ounce and a half by the tenth day.

The next question concerns the sort of food to be given. There is no food so valuable for these infants as breast milk, and every effort should be made to secure this. If the infant is too feeble to suck from the nipple, the milk must be drawn off and given by spoon or pipette. If the baby is capable of sucking from the breast, test-weighing must be carried out to make sure that sufficient nourishment is being given; but if the amount is insufficient, on no account should the baby be taken from the breast, for the value of even partial breast feeding is immense. Any deficiency should be made good, if possible, by the milk of another woman, but if this is unobtainable supplementary feeding may be given with one of the preparations of cow's milk about to be mentioned.

When resort has to be made to artificial feeding, it is seldom possible to advance the diet so quickly as when breast milk is available. Cow's milk needs to be modified in such a way that the protein is either reduced in amount, or predigested by peptonisation, or rendered more easily digestible by acidification. The fat also needs to be reduced, while additional sugar is required to bring the percentage up to that of breast milk. There are various ways by which a feed with these characters may be prepared. One of the most successful is lactic acid milk prepared in the manner described on p. 78; it should be diluted with equal parts of water, and then sugar in the form of dextri-maltose should be added, allowing one teaspoonful to every three ounces of the lactic acid milk and water mixture. Peptonised milk (for method of preparation see p. 79) is sometimes of service, particularly in very small infants. The milk should be diluted with an equal part of water and then peptonised for a full half-hour at first, sugar being added as in the case of lactic acid milk. After the first ten days, the time allowed for peptonisation should be gradually reduced, until by the end of a month it can be left off altogether.

Another milk preparation which is often useful is sweetened condensed milk, owing to its low fat and protein content and relatively high sugar value. In the first few days it should be made with water in a strength of 1 : 10, and then increased to 1 : 8. Because of its low concentration of protein and fat it should only be used as a temporary diet ; when the weight has risen to seven pounds it should be replaced by a half cream or humanised brand of dried milk. For the less severe cases of prematurity, when the weight at birth is as high as five pounds, it is often possible to start feeding with a half-cream dried milk, or an unsweetened form of condensed milk such as Libby's Evaporated milk or Nestlé's "Ideal" brand. If the latter are used they should be diluted with water in the proportion of one part of milk to four parts of water, and a level teaspoonful of sugar should be added to every four ounces of the reconstituted milk.

The tendency of premature infants to become anæmic and rachitic has already been mentioned, and steps must be taken to prevent these conditions. The anæmia may be combated by giving a grain of iron and ammonium citrate [1] in two feeds a day, beginning at the end of the first month and continuing for nine months. The vitamin D requirement of premature babies is greater than that of full term infants, owing to their relatively more rapid rate of growth. They should receive 1,200–1,400 international units daily, throughout winter and summer. This is equivalent to four teaspoonfuls of cod liver oil, which many infants would be unable to tolerate, and one of the concentrates, such as Radiostol, Adexolin, Ostelin, 7 drops twice a day, should be given instead. Administration should begin at one month, and at the same time vitamin C should be commenced, beginning with a few drops of orange juice, and quickly working up to two teaspoonfuls daily ; should this be poorly tolerated, a 25 mgm. tablet of ascorbic acid may be given daily in its place.

Infection probably accounts for nearly half the deaths of premature infants, who have but little power of resistance, and prevention of infection is therefore of the greatest importance. In addition to the strictest asepsis at the time of birth, and

[1] Hæmolac consists of half cream Cow and Gate to which iron and ammonium citrate has been added.

scrupulous cleanliness in the ordinary details of nursing, those in charge must be free of any superficial skin sepsis as well as any respiratory catarrhs. The mother, doctor and nurse should wear a face-mask when handling the baby to prevent the possibility of droplet infection, and fresh masks should be used each day. Visitors should be firmly discouraged, and close admiration of the baby by relatives must be prohibited.

A watch must be kept for cyanotic attacks, which may arise from overfilling of the stomach, from atelectasis, or from cerebral injury. Should these occur, the infant must be handled with the utmost gentleness. Feeds should be weakened for a day or two, but the total amount of water must not be reduced. Inhalation of oxygen with 5 per cent. of CO_2 is invaluable, and is most satisfactorily administered through a nasal catheter, the mixture of gases being bubbled through warm water. Inhalations should be given for ten minutes in each hour. A warm mustard bath may also be used to counter attacks of collapse, and gentle artificial respiration may at times be necessary.

CHAPTER IV

THE DIET OF THE HEALTHY INFANT

Introduction. No one will doubt that the most suitable food on which an infant can be reared is the one designed by Nature, namely breast milk, but unfortunately it often happens that breast milk is not available, and then some substitute has to be employed. Almost always the substitute is cow's milk, but its composition differs from that of breast milk, and so it is generally necessary to modify it with the object of making it as easily digestible as is breast milk. This can be accomplished in so many different ways that the student who seeks one simple and reliable method is likely to think the subject of infant-feeding most complex. It is well for him to remember that no one variety or brand of cow's milk, whether fresh, dried or condensed, transcends all others, and there is no doubt that perfectly healthy babies can be brought up on various systems of cow's milk feeding. In practice it is found that digestive disturbances and nutritional failures in infancy are due less often to defects in the composition of the milk than to errors in the amount allowed, or to faults in the actual technique by which the food is given.

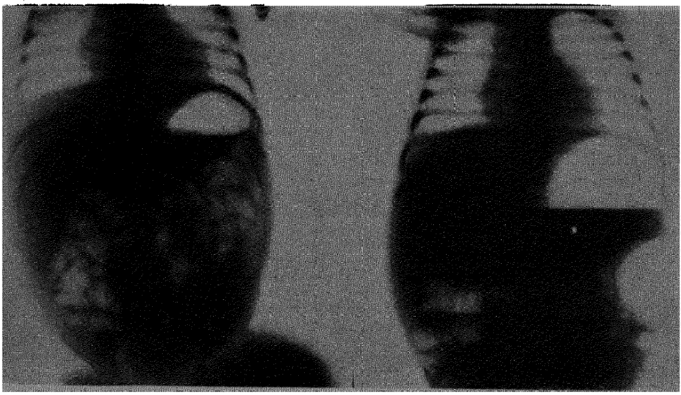

FIG. 6. To show the bubble of air in the stomach. On the left, a normal infant before feeding, and on the right, the same infant after feeding.

In this chapter the diet of healthy infants will be dealt with, leaving to the next chapter the consideration of the management of infants whose digestion is for one reason or another at fault. In the first place certain physiological aspects of the subject may be mentioned.

Sucking. The mechanism of sucking is admirably adapted to the purpose of taking in fluid, and is brought about by the combined action of the tongue, cheeks, and palate. The lips play only a small part in the process, which accounts for the fact that sucking is generally performed quite well by babies who have a hare-lip, while on the other hand a cleft-palate may make sucking impossible. It is a normal happening for a baby to swallow air into the stomach during the act of sucking, and after a meal the stomach generally contains a large bubble of air. The common practice of sitting a baby up at the end of its meal is necessary to facilitate the escape of this air. Excessive swallowing of air, or failure to "break the wind" after a feed, may account for much discomfort and actual dyspepsia; so also did the old practice of wrapping a long binder tightly round the infant's abdomen, preventing the natural expansion at the end of a meal.

Digestive Juices. The various digestive juices are all present in the infant, and the mechanism for digesting the main components of food is ready at birth. Although starch-splitting ferments are present at this age, experience shows that young infants are likely to suffer from excessive fermentation in the intestine if starch is given, leading to intestinal colic and the passage of green frothy stools. It is generally wise to omit starch from the diet until the infant has reached five or six months of age.

Character of Stools. After the first four or five days, when the passage of meconium ceases, the stools of a breast-fed infant should be soft, yellow, and slightly acid to litmus. Two or three should be passed during the day. Infants fed on cow's milk should pass stools which, although more variable in consistence, are formed and yellow, with a slightly alkaline reaction. Curds, which are not present in a healthy stool, appear as yellowish-white plaques generally about as large as a split pea. Almost always they are composed of fat, and are soluble in ether and will float on water. Occasionally protein curds are passed; they are larger, tougher, of a brownish colour, and do not dissolve in ether.

When an infant has diarrhœa the stools are sometimes green, the colour being due chiefly to excessive oxidation in the intestine by bacterial action. Sometimes it happens that stools which are yellow when first passed turn green later on, and this may be confusing unless the colour of the fresh stool is carefully noted. A green frothy stool containing bubbles of gas is a sure indication of too much carbohydrate in the diet. Excess of fat in the diet leads at first to the passage of large pale dry stools, but if the excess continues they become loose and sour-smelling.

Amount of Food required. Many of the troubles of infant nutrition arise because the baby is receiving either too little or too much food, and therefore it is of great help to have some simple means of determining the amount of food that a baby requires. When dealing with healthy babies it is better to be guided by the weight than by the age. The fallacy of relying upon the age will be realised by considering the case of a healthy newborn baby. The birth weight may be anything between 6 and 10 lbs., and yet the 10 lbs. baby would certainly not be satisfied with an amount which would satisfy the 6 lbs. baby. A safe rule is that a healthy infant needs in the twenty-four hours two and a half ounces of fluid for each pound of body-weight, the fluid being either breast milk or some suitable substitute. The total amount of food for the day, determined in this way, must then be divided into the number of feeds that the baby is to receive. Feeding every three hours (6 a.m., 9 am., 12 noon, 3 p.m., 6 p.m. and 10 p.m.) means six feeds a day, four hourly feeding (6 a.m., 10 a.m., 2 p.m., 6 p.m., 10 p.m.) means only five feeds, so that obviously a baby fed every four hours will need more at each feed than if he is fed every three hours. To take an example, an infant weighing 12 lbs. will require $12 \times 2\frac{1}{2} = 30$ ounces of fluid in the day. If he is being fed every three hours he should have five ounces at a feed, but if he is fed every four hours each feed should be six ounces. This is the full allowance ; not all infants will take the full feed, and if such be the case the last half ounce should not be forced. Of course the strength of the food is equally important, but that aspect will be dealt with later, when the various modifications of cow's milk are considered. It must also be borne in mind that owing to their relatively large skin surface infants lose fluid easily, and in hot weather especially they may become fretful owing to thirst. An ounce or more of boiled water should then be given between the regular feeds even if it means that the allotted amount of 2½ ounces of fluid per lb. of body-weight is exceeded.

Other methods are used to calculate the amount of food for an infant, but they lose the advantage of simplicity. For instance, a healthy infant requires in twenty-four hours approximately fifty calories for each pound of body-weight, and a diet may be built up to satisfy the number of calories required. This method demands a knowledge of the calorific value of the various milk preparations, and at any rate in this country the large number of proprietary infant foods makes such a method unwieldy—and one has still to take into account the amount of actual fluid which the baby needs.

The calculation of the amount of food for a weak and wasted baby is a different matter. Here the actual weight would be misleading, and the calculation should be based either on the expected weight or else by using the age of the infant. If the age is used, a useful guide is that so long as an infant is fed *three hourly* he may take up to one ounce more at a feed than his age in months. For instance, an infant of four months may take up to five ounces at a feed, provided he is fed three-hourly.

Times of Feeding. On the first day after birth the infant should be put to the breast every six hours and be allowed to suck for about ten minutes ; on the second day he should feed every four hours ; after that it is generally best for the baby to be fed every three hours, at any rate for the first two or three months. Night feeding is unnecessary for healthy babies, and the habit of going from 10 p.m. until 6 a.m. should start at birth. Although some babies can be trained from birth to take their feeds at four hourly intervals, it is not a good working rule because it so often leads to underfeeding, and, in the mêlée of dyspeptic symptoms which follow, the original cause—that of feeding at too long intervals—is likely to be overlooked. As a general rule the most successful plan is to feed an infant every three hours (six feeds a day) for the first two or three months, and then to change over to four-hourly feeding.

Before leaving this subject, the importance of regularity in feeding must be emphasised. A mother will sometimes state that she feeds her infant whenever he cries, presuming that his crying is due to hunger. It then becomes well-nigh impossible to tell how much nourishment the infant is receiving, and more often than not this sort of practice leads to digestive disturbances. Of course a hungry infant will cry, but the proper management is to increase the amount at each meal, not to feed indiscriminately. A baby should have his meals at regular intervals and if he is asleep at feeding time he should be roused sufficiently to suck. Then occasionally a mother will time the next feed from the end of the previous one instead of from the beginning. Feeds vary considerably in the time taken, one meal being over in ten minutes, another taking half an hour or more, so that this way of timing the feeds leads to much irregularity. The interval should, of course, be measured from the beginning of one meal to the beginning of the next.

Weighing. The best guide to an infant's progress is afforded by regular weighing, which should be recorded once a week during the first six months, and once a fortnight from then until the first birthday. More frequent weighing may lead to needless anxiety owing to the normal daily fluctuation in weight. A satisfactory weekly gain is four to six ounces. So long as the weight rises satisfactorily, and the infant is contented, there need be no concern about his progress.

BREAST FEEDING

As an infant food, breast milk has several advantages over other milks. Its composition is well adapted to the physiological requirements of the infant's digestion, as a rule it contains an adequate supply of vitamins, and it possesses certain protective antibodies of specific value to the growing infant. It is also given at a uniform temperature, and has the great merit of being clean. Breast milk is often a deciding factor in the survival of a premature infant or one enfeebled by disease.

Composition of Breast Milk. For the first few days after birth the breasts secrete a thin yellow slightly alkaline fluid called *colostrum*, which is relatively richer in protein and poorer in fat and sugar than breast milk. Holt gives the percentage composition of colostrum during the first two days after birth as follows :—

Percentage Composition of Colostrum

Protein.	Fat.	Lactose.	Ash.	Water.
8·6	2·3	3·2	0·3	85·6

Microscopically colostrum is characterised by the presence of large cells which are filled with fat granules—colostrum corpuscles. Colostrum is of value to the baby as a means of introducing food in a very easily digestible form, and it is also thought to transmit in its high protein content antibodies and protective substances from the mother to her infant. The secretion of colostrum lasts for about a week to ten days. From then until the end of the first month the milk gradually attains its mature proportions, which it then maintains practically unaltered for about nine months.

Average samples of breast milk taken between the first and ninth month after birth show the proportion of the main constituents to be as follows (Holt) :—

Percentage Composition of Breast Milk.

Protein.	Fat.	Carbohydrate.	Ash.	Water.
1·25	3·5	7·5	0·2	87

The caloric value is approximately twenty calories per ounce.

The protein is in two forms, lactalbumin and caseinogen, the former being present in approximately twice the amount of the latter ; this is a state of affairs which is more than reversed in cow's milk, in which the caseinogen is from three to five times as plentiful as the lactalbumin. The casein curd of human milk is produced by the conversion of soluble caseinogen into insoluble casein by the action of rennin in the presence of calcium salts, and is a fine small curd, differing very much from the heavy curd of cow's milk. The fat of breast milk is present in a fine emulsion, and the carbohydrate is entirely in the form of lactose. Of these three chief constituents, the fat is subject to slight variations in amount, while the sugar is the most constant.

Of the mineral salts, iron is present only in small amounts in both human and cow's milk. The iron content of breast milk is about 0·16 mgm. per cent. This is insufficient for the needs of the growing infant, and during the early months the reserve of iron which has been stored in the liver during fœtal life is drawn upon—as is also the store of copper. This satisfies the infant's requirements for about six months, but unless iron in some form, such as meat juice or meat and vegetable broth, is then added to the diet, a hypochromic type of anæmia is very likely to appear (nutritional anæmia). Provided that the mother is healthy, getting out of doors every day, and taking a diet which includes an ample supply of vitamins, the vitamin content of her milk is adequate for the child's needs.

Variations in Quantity. The amount of breast milk which the baby obtains is estimated by test-feeds, that is to say by weighing the infant at the beginning and again at the end of a feed. The difference in the weights indicates the amount of milk which the baby has drawn off. While the test-feed is being carried out the napkins must not be changed, because any urine or fæces voided during the meal must be included in the second weighing. Test-feeds are of the greatest help in deciding whether the infant is getting sufficient nourishment from the breast, but as the amount of breast milk at consecutive feeds may vary by as much as two or three ounces, weighings taken only at one meal are of little value. It is essential to test-feed at each meal over a twenty-four hour period ; it is then possible to arrive at the *average* amount of milk per feed. The first feed of the day is generally the largest. Tallerman and Hamilton,[1] from a series of analyses, concluded that the midday breast feed was only slightly more than half the early morning feed, while the last feed at night tended to be a little greater than the midday feed.

Not only does the secretion of breast milk vary during the day, but also, of course, with the age of the infant. While only an ounce is secreted at each feed during the first week, by the third month the average secretion per feed has risen to four ounces, and at the sixth month to six or seven ounces. The amount also varies with the stimulus of sucking, for Budin [2] has shown that the amount of a wet-nurse's milk increases with the number of infants she is suckling.

Domestic cares and worries have a definitely adverse influence on the supply of breast milk. This is particularly obvious in hospital practice, where one so often is told that the breast milk failed as soon as the mother " got up ", which meant returning to a full day's work. In order to keep up the supply of breast milk, a rest in the middle of the day for an hour or so is important. The supply is likely to dwindle if the mother is worried because her baby is not getting on, and the more she worries the less milk she has to offer the infant. It is essential to restore the mother's confidence, and often a short spell of supplementary feeding may be the best way to do this, for as the baby begins to go ahead the mother ceases to worry and her breast flow increases. Efforts to increase the amount of breast milk include light massage of the breasts, and douching them alternately with hot and cold water. The use of galactagogues such as " Lactagol " —of which a teaspoonful may be taken thrice daily—is occasionally helpful, but more often is disappointing.

Variations in Composition. The fat is the most variable constituent of breast milk. At the midday and afternoon feeds it may be as much as 2 per cent. above the amount in the early morning feed, and not only does it vary at different meals, but it also varies during the meal. The milk taken at the end of a feed (the strippings) may contain three or four per cent. more fat than a sample at the beginning of a meal. The practical importance of this is that if the infant is to derive the full benefit from the milk he must empty one breast properly before being put to the other breast. Mothers often feed their infants for ten minutes from each breast ; this is satisfactory provided that ten minutes suffices for the infant to empty each breast, but otherwise it is better to advise that alternate breasts are properly emptied at consecutive feeds, the other breast then being used to make good any deficit. The variation in fat must also be remembered when a sample of breast milk is taken for analysis. Either the whole content of a breast should be analysed, or the infant may be allowed to suck for about five minutes before taking a sample. This will approximate to the middle of the feed, and analysis will then give a fair idea of the strength of the milk.

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[1] Tallerman and Hamilton, " Infant Nutrition," London, 1928.

[2] Budin, " The Nursling," Paris, 1907.

There is a tendency during the nine months of lactation for the protein to diminish in amount and the lactose to increase, but these changes are too small to be of importance.

Unless the breast milk is of poor quality, it is very difficult to affect its composition by altering the mother's diet. A deficient maternal diet simply leads to a reduction in the total output of milk rather than of any particular constituent. It is unnecessary for a nursing-mother to take extra quantities of milk and rich foods; it is more important for her to have a well-balanced diet containing all the ordinary foods and with a liberal amount of fluid. Milk, butter, eggs, meat and fresh vegetables should all be included for their vitamin value, but an excess of them is not called for.

It is well to remember that certain drugs given to the mother may be excreted in the milk, and so may affect the child. Cathartic salts, belladonna, arsenic, the salicylates, iodides and bromides may reach the infant in this way.

Technique of Breast Feeding. Efficient breast feeding should be a quiet and comfortable process for both mother and baby. The mother should sit on a low chair, holding the baby in the crook of one arm so that the other hand is free to manage the nipple. The infant's face must not become buried in the breast, since this would prevent him from breathing comfortably through his nose ; lack of attention to this detail will account sometimes for a mother saying that she cannot feed her baby because he keeps turning away from the breast.

A breast feed generally lasts about twenty minutes. An infant will take all it requires in that time, although some infants if allowed to do so will suck for three-quarters of an hour or more. Most of this time they are not really sucking, but merely mouthing the nipple. Consecutive feeds should always begin on alternate breasts, making sure that one breast is properly emptied at each feed, since incomplete emptying of a breast will lead to a gradual diminution of the amount of milk secreted. The decision to give one or both breasts at a feed depends simply upon the amount of milk produced.

At the beginning and end of the feed the nipple should be swabbed with weak boracic lotion, because although breast milk is to all intents and purposes sterile, if there are any bacteria they will come from the nipple and the openings of the larger ducts.

Contra-indications to Breast Feeding. *Diseases of the Mother.* The appearance of any of the acute infectious fevers in the mother will necessitate weaning. Chronic nephritis also excludes nursing, not that the milk is detrimental to the infant, but because the extra strain thrown on the maternal health is harmful. Eclampsia is another contra-indication. A mother who is suffering from pulmonary tuberculosis must not be allowed to suckle her infant, partly because after parturition the pulmonary condition is very likely to light up, and also because there is a very real risk of the baby becoming infected with tuberculosis—not through the mother's milk unless the breast is also tuberculous, but by direct inhalation from her. When the mother is suffering from malignant disease it is as a rule wiser to wean the infant.

Breast feeding may safely continue after the return of the menstrual periods, but if another pregnancy begins during lactation it is better to wean the baby, so that there may be no extra calls upon the mother beyond those of the fœtus.

Diseases of the Breast. Severe retraction of the nipple may make ordinary sucking impossible ; as much as possible of the milk should then be drawn off from the breast with a pump. Fissure of the nipple generally makes sucking so painful as to necessitate weaning. It is best treated by bathing several times a day with boracic lotion and then either cauterising the fissure with silver nitrate or painting it with Friar's Balsam. A fissure can usually be healed in about a week and then the infant may return to the breast, but it will be necessary to protect the part with a nipple shield. When suppuration occurs in the breast, weaning is, of course, necessary. It is often taught that an infant may continue to suck from the sound breast when there is an abscess in the opposite breast, but this is a very unwise practice, as the baby is very likely to become infected. At best, the milk from the unaffected breast should be drawn off and boiled before it is given to the infant. Malignant disease and tuberculosis of the breast are absolute bars to breast feeding.

Disease of the Infant. The deformities of hare-lip and cleft-palate may be sufficiently severe to prevent suction either from the breast or a bottle. The breast milk must then be drawn off and given to the infant from a spoon or a pipette. It is difficult under such circumstances to maintain the supply of breast milk without the natural stimulus of the baby's suction, and resort generally has to be made to artificial feeding.

Inadequate Reasons for Weaning. One or other of the above indications is not as a rule the reason of premature weaning, more often one is told that " the milk did not agree with the baby ", or " the milk gave the baby such a lot of wind ". Generally these disturbances could have been quickly remedied without recourse to weaning, and in most cases they can be traced to one of the three following causes : Faulty technique of feeding, overfeeding, or underfeeding. It is very exceptional for the composition of the milk to upset the baby, and to wean a child prematurely from a mother who can still produce milk is often to court disaster, for an undiscovered fault in the technique of breast feeding is likely to continue in bottle feeding, and also there may be a real difficulty in finding a suitable artificial food.

Faulty technique of feeding. In this category are included those infants who turn away from the breast because they are held too closely and are partly smothered. Congenital enlargement of the adenoids, and snuffles associated with catarrhal rhinitis or with syphilis, may make nose breathing difficult and interfere with sucking, but in these cases the difficulty of sucking is just as great from a bottle as from the breast. Two drops of liquid paraffin instilled into the anterior nares a minute or two before a feed is due will help to make the breathing easier just while the meal is in progress.

Another technical fault may be that the feeds are spaced too far apart, usually at four-hourly intervals, with the result that the baby is unduly hungry at meal times ; he then sucks too greedily and gulps down large quantities of air with the milk. The weight ceases to rise, and there is much flatulence with attacks of colic and screaming. All that is needed is to reduce the times of feeding from every four hours to every three hours. Half an ounce of boiled water at the beginning of a feed will tend to keep the baby from sucking too greedily, and a carminative mixture after the feed will often prove useful. Some such prescription as the following may be given :—

℞ Sodii Bicarbonatis gr. iiss.
Spiritus Ammoniæ Aromaticus m. iiss.
Tincturæ Cardomomi Compositi m. iiss.
Aquam Anethi ad ℥i.

The addition of chloral gr. ½ will help the infant to settle off to sleep after the feed instead of tossing fretfully.

Overfeeding. This is an uncommon fault. The infant is likely to regurgitate some of the milk at the end of the feed, and although at first the gain in weight is rapid, and the baby seems to be doing very well, the stools are large and frequent, and later become loose, curdy and green, while the weight no longer rises. Test feeds should always be carried out before making the diagnosis. The correct management is to lengthen the interval between the feeds, and to limit each feed to the contents of one breast. A little boiled water before feeds will also help these cases, partly by curbing the baby's appetite, and also by diluting the milk. Occasionally the mother is found to be taking an unnecessarily large diet with the intention of maintaining a good supply of milk.

Underfeeding. This is much more frequently met with than overfeeding. The history usually is that the baby sucks vigorously for a few minutes before turning away from the breast, or he may go on sucking for some time after the breast is empty, swallowing a good deal of air, which leads to attacks of colicky pain. Under such circumstances the gain in weight is negligible, the bowels are likely to be constipated, and the motions are small. Test feeds over a period of twenty-four hours will at once indicate the degree of underfeeding. Treatment consists in giving both breasts at each feed, and care must be taken to see that the infant is not allowed to continue sucking after the breast is empty. If, in spite of feeding from both breasts every three hours, test feeds still show a deficit, it must be made good by supplementary feeding.

Supplementary Feeding. Supplementary feeding may be carried out either by replacing one or two breast feeds entirely by bottle feeds, or by giving the baby a small amount from a bottle after each breast feed. The supplementary feed should never be given before the breast feed, otherwise the infant may soon come to prefer the bottle and refuse the breast. The first method is the more simple, a bottle being given in place of the midday and 6 p.m. breast feeds. If it is necessary to replace three breast feeds, this would be done at 9 a.m., 3 p.m. and 6 p.m. The better method is to supplement each breast feed except the early morning feed, because then the breasts will more frequently receive the stimulus of sucking. At each feed the baby should first of all empty both breasts, which he will do in about a quarter of an hour. The amount of supplementary food required must then be gauged by test feeding. Test feeds need not be done at every meal, it will be sufficient to test feed over twenty-four-hour periods twice a week. The progress of the infant's weight will give the surest indication of whether enough of the supplementary food is being given.

It is a mistake to make the supplementary food too sweet, as the infant may then prefer it to the breast milk. Two parts of milk to one part of water with a teaspoonful of sugar to every four ounces does very well, or a half-cream dried milk, prepared by adding a drachm of milk-powder to an ounce of water, may be used instead.

Wet Nursing. In the case of a premature or feeble baby, breast milk is often essential, and if the mother is unable to nurse her infant, the services of a wet nurse may be invaluable. When a wet nurse is chosen she must undergo a preliminary examination to be sure that her health is good. Tuberculosis and syphilis must be excluded, and a Wassermann reaction should be performed. The wet nurse's own infant should also be examined, as evidence of syphilis may be found even when examination of the mother has failed to do so. The wet nurse should be allowed to keep her own infant with her, for if her supply of milk is to remain satisfactory she must be protected from unnecessary anxiety—such as separation from her baby would entail.

Seeing that human milk does not vary in composition between the first and ninth months, it is not necessary that the wet nurse's infant should be of the same age as the foster-child, and by employing a wet nurse whose infant is at least three months old, one can judge the effect of her milk upon her own child. But however sturdy her own infant may be, there can be no guarantee that the milk will suit the foster-baby. Debbas[1] has recorded an infant who was fed from its mother for three months, and then the milk of a wet nurse was substituted, but with the first two feeds after the change the infant suffered from such severe anaphylactic shock that the services of the wet nurse had to be discontinued.

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[1] Debbas, E., *Paris Médicale*, 1926, **59**, 506.

ARTIFICIAL FEEDING

When breast milk is not available, and resort has to be made to artificial feeding, almost invariably cow's milk, or some modification of it, has to be used. A knowledge of the comparative composition of human and cow's milk is therefore essential to the study of the artificial feeding of infants.

Composition of Cow's Milk. Fresh cow's milk should be amphoteric or slightly acid. Its composition is shown in the following table :—

Percentage Composition of Cow's Milk (Holt)

	Protein.	Fat.	Lactose.	Ash.	Water.
Cow's milk . .	3·5	3·5	4·75	0·75	87
Human milk . .	1·25	3·5	7·5	0·2	87

These figures apply to the milk of most breeds of cattle, but the milk of Jersey cows is richer in all constituents, especially fat, which may be as high as 5·5 per cent. The caloric value of cow's milk is approximately 18 calories per ounce.

Compared with human milk, cow's milk is more than twice as rich in protein. The proportion of caseinogen to lactalbumin is also very different in the two milks, for while in human milk there is twice as much lactalbumin as caseinogen, cow's milk contains from three to five times as much caseinogen as lactalbumin. When this high content of caseinogen reaches the infant's stomach a large and tough curd is produced, which accounts for some of the difficulty young infants may experience in digesting cow's milk.

The amount of fat in cow's milk is the same as in human milk, and if the milk is collected from a mixed herd the percentage of fat shows little variation, but it is not in so fine an emulsion, which makes its digestion more difficult. The amount of lactose is considerably less than in human milk, and so additional sugar is required. In many of the proprietary preparations of dried cow's milk this addition of sugar is made by the manufacturers (see pp. 63, 64).

The mineral salts are present in sufficient amount for the infant's requirements, calcium, sodium, and potassium salts being particularly plentiful. The proportion of iron is, however, very small—0·06 mgm. per cent.—which is even less than in human milk. It has already been pointed out that breast-fed infants require iron in some form added to the diet after the sixth month, and this is equally necessary for infants reared on cow's milk.

Fresh cow's milk contains all the various vitamins, but their concentration is subject to variation. For instance, cow's milk during the summer is richer in anti-rachitic vitamin than during the winter, when often the animals are entirely stall-fed, and so are not exposed to the sun's rays. It is also important to bear in mind that one of the effects of heating cow's milk is to destroy its content of vitamin C.

Grading of Cow's Milk. The value to the community of a milk supply which is safe from bacterial contamination cannot be exaggerated. That the milk should be free from infection with tubercle bacilli is perhaps of greatest importance, but pathogenic organisms or spores of any sort ought not to be present. The necessity of a bacterially clean milk supply has been officially recognised since the Milk and Dairies Act of 1922. The Milk Order of 1936 has substituted the three following classes of milk for the previous grades.

1. *Tuberculin-tested Milk.* This milk is drawn from herds which have to be submitted to tuberculin tests every six months. The milk, which before delivery must not be heated, may either be bottled on the farm or by the dealer, and the bottle must be closed with a tight disc and covered with a suitable outer cap to make a complete seal, and bear on it the date of milking. This milk must not contain more than 200,000 bacteria per c.c., nor any coliform organisms in 0·01 c.c. The price of milk prepared under these precautions is unfortunately about twice that of ordinary milk.

A special licence is required for the sale of Tuberculin-tested Milk which has also been pasteurised. The bottle must bear the designation "*Tuberculin-tested Milk* (*Pasteurised*)."

2. *Accredited Milk.* This milk is drawn from cattle which are submitted to a veterinary inspection every three months. No cows which, to the knowledge of the producer, have reacted positively to tuberculin tests may be included in the herd, but routine tuberculin tests on the herd are not performed; otherwise the bacterial content conforms with the milk in the previous class.

3. *Pasteurised Milk.* In this grade the milk before delivery is heated to 145° to 150° F., for half-an-hour, and then is quickly cooled to a temperature not higher than 55° F. The bacterial content of this milk must not exceed 100,000 organisms per c.c.

Sterilisation of Cow's Milk. Even the finest grades of fresh cow's milk possess a bacterial content considerably in excess of breast milk, and therefore before cow's milk can be regarded as a suitable food for infants it is necessary to carry out some form of sterilisation. The two methods by which cow's milk can be sterilised in the home are by boiling and by pasteurisation. When boiling is the method selected, it is sufficient to heat the milk until it rises in the saucepan, or a better method is to warm the milk in a double saucepan until bubbles are rising to the surface. This corresponds to a temperature just below boiling, and the milk should be kept at this temperature for two or three minutes before cooling.

Pasteurisation implies raising the milk to a temperature of 155° to 160° F., and keeping it at that temperature for twenty minutes to half an hour before it is allowed to cool. There are several different patterns of apparatus for effecting pasteurisation, but the principle is the same in all, the milk being put into bottles which are heated in a water container for the requisite time.

It is essential that sterilisation should be done sufficiently to destroy the tubercle bacillus, and this is accomplished either by raising milk to the boil or by pasteurisation. Other pathogenic organisms are killed at a slightly lower temperature than the tubercle bacillus, for instance streptococci are destroyed at 65° C. (150° F.), while *B. coli* and the organisms of dysentery, typhoid, cholera, diphtheria and malta-fever, are killed by boiling or by maintaining the milk at 60° C.(140°F.) for twenty minutes. There is no doubt that pasteurisation of milk makes it a perfectly safe infant food, and actually most of the milk consumed in London is pasteurised before delivery. It has always seemed to the author that in a private house the boiling of milk is not only carried out more quickly and simply than pasteurisation, but is also cheaper, and therefore is the method of choice.

Both boiling and pasteurisation produce other effects on milk besides sterility, and from this point of view there is little to choose between the two methods. The casein curd of heated milk is a little easier to digest than the curd of raw milk because it is rendered finer and softer, while if the milk is brought to the boil the lactalbumin coagulates and accounts for the thin skin which forms on the top of the milk. The fat and the lactose are unaltered unless the milk is heated above boiling, when caramelisation of the sugar may give the milk a rather unpleasant flavour. With regard to the vitamins, the fat-soluble vitamins A and D are heat-stable, but the anti-scorbutic vitamin C is largely destroyed, and therefore when an infant is fed on a milk which has been sterilised by heating, it is essential to replace this vitamin by giving fresh fruit juice.

The only disadvantages of sterilising milk are the destruction of vitamin C, and a slight alteration in the taste ; there is no evidence that sterilisation by heat diminishes its nutritive value. The advantages are that the risk of tuberculous infection is avoided, as is the chance of other bacterial contamination, and the protein is rendered easier to digest. The advantages easily outweigh the disadvantages, and so long as the necessity of replacing vitamin C in the diet is borne in mind, it should always be recommended that cow's milk intended for infant feeding should be sterilised by one of these two methods.

Technique of Bottle Feeding. It is important that attention should be paid to the small details of bottle feeding, for it is surprising how often a mild digestive disturbance can be traced to some simple error in the way the feed is given. When the milk is pasteurised in a private house, it is more convenient to prepare at one time enough bottle feeds to last the whole day, then merely warming a bottle at each feed. Otherwise each meal should be prepared separately. After the feed has been given, the bottle and the rubber teat and valve should be washed thoroughly in boiling water and then be placed in a dish and covered with water which has been boiled, remaining there until the next feed is due.

Care must be taken to see that the hole in the teat is the right size. This can easily be gauged by shaking the bottle, when if the hole is the correct size each shake should produce one drop of milk from the teat. If the hole is too large the meal is taken very quickly, and a good deal of air is gulped down ; noisy eructations follow, some of the milk is regurgitated as well, and colicky attacks are a common sequel. If the hole is too small the meal may become an interminable affair, and the infant may give up sucking before the bottle is emptied. A failure to gain weight is the inevitable result.

When the feed is actually in progress the infant should be out of his cot, and propped up on the mother's arm, as this facilitates swallowing and prevents regurgitation. The practice of propping the bottle on a pillow within reach of the baby's mouth, so that the infant can continue his meal unattended, is a thoroughly bad one. The bottle must, of course, be kept tilted so that the teat is always filled with milk, otherwise a good deal of air will be swallowed.

Whole-milk Feeding. Although infants will sometimes thrive satisfactorily if they are reared from birth on undiluted cow's milk which has simply been sweetened, it is not a method of feeding that can be confidently recommended. The greater concentration of caseinogen and the more coarse emulsion of the fat of cow's milk as compared with breast milk call for some preliminary adjustment before cow's milk can be safely depended upon as an infant food. Undiluted cow's milk should seldom be used below six months of age, but from then onwards there is seldom any need to continue to dilute it.

Diluted-milk Feeding. The purpose of diluting cow's milk is simply to render it more easily digestible, particularly with regard to its casein and fat content. In addition to diluting the milk, enough sugar must be added to bring the carbohydrate value up to that of breast milk, but it must be understood that no amount of manipulation of cow's milk can in truth " humanise " it.

Degree of dilution. After the end of the first fortnight it is unnecessary to dilute cow's milk below a strength of two parts of milk to one part of water. If an infant cannot tolerate this strength of milk and water it is better to give one of the proprietary dried-milk foods than to give weaker dilutions of fresh milk, for this will almost certainly lead to severe underfeeding. During the first fortnight the milk should be diluted with an equal quantity of water ; from then until the fourth month the amount of milk should be double the amount of water, and from the fourth to the sixth month the mixture should be enriched to three parts of milk and one of water. After the sixth month dilution is seldom needed, and a healthy infant can then be given undiluted milk. The changes in the strength of the mixture should not be made suddenly, but should be carried out gradually by altering the strength of one feed in the day to start with, and taking about a fortnight before all the feeds in the day have been strengthened.

The following table shows the proportion of milk and diluent that should be used until the ninth month, and also gives the percentage composition of the resulting mixture :

Period.	Proportion of Milk.	Proportion of Water.	Protein (Per cent.).	Fat (Per cent.).	Lactose (Per cent.).
0–2 weeks . .	1	1	1·75	1·75	2·4
2 weeks–4 months.	2	1	2·3	2·3	3·2
4–6 months . .	3	1	2·6	2·6	3·5
6–9 months . .	Whole milk	—	3·5	3·5	4·75

Addition of Sugar. A level teaspoonful of sugar when added to a three-ounce feed increases the sugar by roughly 5 per cent. Of course, such measures cannot be exact because domestic teaspoons vary in size, and the density of the various sugars differs so that a level teaspoonful of one will weigh more than another. Fortunately the digestive capacity of a healthy baby is somewhat elastic, and scientific precision in the percentage composition of the feed is not absolutely essential to the well-being of the baby. To mixtures of equal parts of milk and water, or two parts of milk and one part of water, add a teaspoonful of sugar to every three ounces ; to stronger mixtures allow a teaspoonful of sugar to every four ounces. If the stools tend to become too formed and hard, an extra teaspoonful of sugar should be added to each feed, while if the stools tend to be relaxed, the sugar should be temporarily reduced.

For a healthy baby it is not essential that the added sugar should be in the form of lactose or sugar-of-milk. Brown demerara sugar (cane-sugar) is generally quite satisfactory, and is cheaper than lactose. Glucose may also be used, its chief advantage being that it requires no digestion at all, and is ready for absorption as soon as it reaches the stomach. If there has been any tendency to indigestion of sugar, as shown by the passage of loose green frothy stools, dextri-maltose (a mixture of dextrins and maltose) will be found a useful form of carbohydrate on account of the small amount of fermentation to which it gives rise. Starch as a form of carbohydrate should not be added until the sixth month.

Addition of Cream. It has already been pointed out that the fat of cow's milk is not so easily digested as the fat of breast milk. Provided that cow's milk is not diluted below a half and half strength, so that the fat content is not lower than 1·75 per cent., it is generally wiser not to attempt to make good the deficit of fat. During the winter months the infant who is reared on cow's milk should be given a teaspoonful of cod-liver oil emulsion twice a day, which incidentally will mean a little extra fat in the diet.

Premature or very feeble infants are on rather a different footing, for it may be necessary at first to dilute their milk weaker than half-milk half-water, and then the addition of fat may be necessary. It can be added as cod-liver oil emulsion or in the form of cream. Cream is generally prepared soon after milking by a machine separator, and contains about 40 to 50 per cent. of fat. Gravity cream, which is obtained by allowing milk to stand for several hours and then collecting the cream which has risen to the top, contains only about 16 per cent. of fat, and because it has stood for some time it is likely to be bacterially contaminated, and so is not suitable for infant feeding. If it is decided to add separated cream, not more than half a teaspoonful should be allowed for every three ounces of the feed. But it may be repeated here that extra cream is both unnecessary and unwise in the diet of full-term healthy infants.

Diluents. Various fluids may be used for the purpose of diluting milk. Unless some other diluent is particularly indicated, water serves perfectly well, and it has the advantages of requiring no preparation and no digestion. Barley water [1] is often used instead of plain water. It contains from 1 to 2 per cent. of starch, and sometimes even this small amount may give rise to looseness of the motions. Use can be made of this when a baby shows a tendency to constipation, the employment of barley water instead of plain water being sometimes sufficient to ensure a daily action of the bowels. Barley water also facilitates the digestion of milk protein, because the starch encloses the small curd particles in a colloidal envelope, and so prevents them from running together to form large curds. It may be mentioned here that the addition of lime-water to a milk-and-water feed (in the proportion of a tablespoonful of lime-water to every three ounces) tends to have a constipating effect on the bowels. To give lime-water with the idea of promoting calcification of the bones and teeth is fallacious, for there is sufficient calcium and phosphorus in the milk to satisfy the infant's requirements in this direction.

Whey [2] is a valuable diluent as it consists of milk from which the caseinogen and part of the fat have been removed, while the lactalbumin, lactose and mineral salts remain. If the curd is broken up before the whey is strained off, the whey will contain about 1 per cent. of fat. A mixture of equal parts of milk and whey differs from whole milk only in containing half the amount of caseinogen and two-thirds the amount of fat. The elements in cow's milk that make its digestion difficult are in this way reduced, and a mixture of milk and whey is an efficient infant-food, but it is not often used because of the amount of preparation required.

[1] *To prepare Barley water.* Put two heaped teaspoonfuls of washed pearl barley to a pint of cold water, boil slowly down to two-thirds of a pint, and strain.

[2] *To prepare whey.* Add one teaspoonful of rennet to a pint of milk. Keep the milk just warm for half-an-hour, by which time a curd will have formed. Break up the curd with a fork, strain off the whey and take it quickly to the boil. Keep the whey in a cool place until required for the feeds.

PROPRIETARY INFANT FOODS

Proprietary infant foods fall into three classes :—

(1) Dried milk.
(2) Condensed milk.
(3) Foods intended only as additions to milk.

Dried Milk. Milk is usually dried in one of two ways : either it is poured on to hot rollers, by which the water is driven off and the milk powder is then scraped off the rollers, or else it is sprayed through a heated chamber where the water is driven off while the solids fall as a powder to the bottom of the chamber. Drying by the latter process keeps the fat in a finer emulsion than does the former method, for when roller-dried milk is reconstituted with water, the fat often separates out as oily globules, but from the point of view of digestibility there is little to choose between the two methods.

Dried milk has the great advantage of being practically sterile, and as the powder will keep for several months it makes a most convenient method of feeding during long voyages, and during residence in hot climates where it may be difficult to obtain fresh clean cow's milk. Even in temperate climates the sterility of dried milk is a great asset, and where the circumstances of the home are such that bacterial contamination of fresh milk seems likely, dried milk is certainly to be preferred. One constant effect of drying milk is the destruction of the anti-scorbutic vitamin, and therefore when dried milk is used for infant feeding it is essential to give fresh fruit juice.

There are several different proprietary preparations of dried milk, but they all fall into one of the two following categories :—

(*a*) Dried whole milk, often spoken of as full-cream dried milk.

(*b*) Dried modified milk, often advertised by the manufacturers as humanised milk.

The milks in both categories are intended to be used instead of fresh cow's milk.

Dried Whole Milk. The table on page 62 gives the percentage composition of some of the more common examples of dried whole milk, after the powder has been reconstituted with water.

From this table it will be seen that the main constituents of the dried whole milks are present in practically the same proportions in each variety. In all of them the amount of sugar is less than that of breast milk, and this should be made good by adding a level teaspoonful of sugar to every four ounces of the reconstituted milk. Needless to say these foods are prepared from the best milk available, and therefore they are rich foods, especially in their fat value, and for this very reason they are poorly tolerated by newborn infants.

Dried Whole Milk (Full-cream Dried Milk).

Brand of Milk.	Reconstituted by adding one drachm of Powder to one ounce of Water.			Remarks.
	Protein.	Fat	Carbohydrate.	
Ambrosia (Full cream)	3·3	3·5	4·6	
Cow and Gate (Full cream).	3·3	3·4	4·7	
Dorsella (Full cream)	3·1	3·4	4·8	
Glaxo (Full cream) .	3·1	3·3	4·8	165 International units vitamin D per pint.
Ostermilk, No. 2 (Glaxo, Ltd.).	3·1	3·3	4·8	
Lacta, No. 1 . .	3·4	3·2	4·6	Lacta, No. 2, contains additional vitamin D and iron.
Trufood (Full cream)	3·9	3·6	5·4	

NATIONAL DRIED MILK. This is a roller-dried full-cream milk introduced by H.M. Government as a wartime measure to replace proprietary dried milks. A measure holding 56 grains is supplied, and a measure of powder to an ounce of water gives a composition equivalent to that of the proprietary brands in the above table, the calorie value being 18 per ounce.

The reconstitution of practically all the dried milks is performed by mixing one drachm of the powder with one ounce of water,[1] and it is necessary to remember that reconstitution at this strength gives a fluid which virtually amounts to whole cow's milk. The caloric value of these reconstituted milks averages 18 calories per ounce, which is the same caloric value as fresh cow's milk, so that they lose nothing in nutritive value as a result of being dried. It has already been pointed out that undiluted fresh milk is too strong for the average infant during his first few months, and similarly the dried whole milks should not be employed during at least the first three months of infancy.

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[1] Importance attaches to the sort of measure which is used in the preparation of these foods. Domestic teaspoons not only vary considerably in size, but also, while a "teaspoonful" will denote to one mother a heaped spoonful, another will barely fill the spoon level, and the resulting milk may be either stronger or weaker than is intended. Generally speaking, a slightly heaped teaspoonful holds about a drachm and half of powder, but this is only a rough guide. Most of the manufacturers of dried milks have appreciated this difficulty, and supply in their tins of food a measure which should be filled just level. Unfortunately the measures of different firms vary in size, for instance the Glaxo measure holds a drachm, the Allenbury measure two drachms, and the Lactogen measure four drachms, so that they are not interchangeable, but if the directions for dilution issued with each tin are followed, the resulting milk will be reconstituted approximately at a strength of a drachm of powder to an ounce of water.

Dried Modified Milk.

Brand of Milk.	Reconstituted by adding one drachm of Powder to one ounce of Water.			Remarks.
	Protein.	Fat.	Carbo-hydrate.	
Allenbury, No 1 .	1·7	3·1	10·6	Intended for infants of one to three months. Some casein has been removed, and vegetable albumin and lactose, dextri-maltose, iron and vitamin D added.
Allenbury, No. 2 .	1·9	3·0	10·4	Intended for infants of three to six months. Similar to No. 1. Contains no starch.
Allenbury Half Cream	2·0	1·5	12·1	Contains added dextri-maltose.
Almata . . .	1·6	3·2	7·3	A much modified milk. The constituents of cow's milk are all present with, in addition, egg-yolk, butter, malto-dextrins, mineral salts, and decitrated fruit juice.
Ambrosia (Humanised)	2·0	2·5	7·2	Contains additional lactose and 200 International units vitamin D per pint.
Ambrosia (Half cream)	2·5	1·8	7·2	
Berina . . .	2·4	2·1	7·0	Dried irradiated milk with added cream of wheat and lactose. *Contains starch.*
Cow and Gate (Half cream).	2·5	1·8	7·2	Contains added lactose.
Cow and Gate (Separated).	3·5	0·1	5·3	The fat has been entirely removed, and the food should only be used as a temporary measure in fat-intolerance
Dorsella (Humanised)	1·5	3·2	6·9	
Eledon (1 in 10) .	2·9	1·4	4·0	Butter-milk, bacterially acidified. One part of powder should be diluted with ten parts of water.
Glaxo [1] (Sunshine) .	2·1	2·5	7·0	Contains added vitamin D. Each ounce of reconstituted milk is said to contain the equivalent of two and a half drops of cod-liver oil.
Ostermilk, No. 1 (Glaxo, Ltd.).	2·1	2·5	7·0	

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[1] *Sunshine Glaxo* is also supplied to Public Health Authorities under the name of "Ostermilk."

Half Cream National Dried Milk. Introduced as a wartime measure to replace the proprietary half cream and humanised brands. When reconstituted by adding a drachm or measure of powder to an ounce of water, the fat content is 2 per cent. Unlike the proprietary brands this milk is unsweetened, and the addition of sugar is therefore necessary.

Dried Modified Milk.

Brand of Milk.	Reconstituted by adding one drachm of Powder to one ounce of Water.			Remarks.
	Protein	Fat	Carbohydrate.	
Glaxo (Three-quarter cream)	3·1	2·5	5·8	
Horlick's Malted Milk	1·8	1·0	8·8	Made by drying together milk and extracts of wheat and barley malt. When made according to directions, it contains no unaltered starch.
Lacidac[1] (Cow and Gate).				
Lacta (Modified, No. 1)	2·5	2·2	6·7	Lacta, modified, No. 2, contains additional vitamin D.
Lactogen (Nestlé Ltd.)	2·0	3·1	6·6	
Recolac (Mead, Johnson & Co.).	2·0	3·3	6·6	Contains added dextri-maltose. The fat is replaced by vegetable and animal fat.
Trufood (Humanised)	1·4	3·45	6·25	Contains added cream and lactose Casein 0·8 per cent. Lactalbumin 0·6 per cent.

Dried Modified Milk. The table on pp. 63–64 shows the percentage composition of some of these foods after the powder has been reconstituted with water.

It will be seen that the amount of protein and carbohydrate has been made to approximate roughly to the proportions present in human milk, and therefore the addition of sugar is not required. In several instances the fat value has been slightly reduced, which probably accounts for the fact that these milks are particularly suitable for the feeding of young infants. When it is intended to rear an infant on dried milk, the principle that is most likely to succeed is to give a half cream or " humanised " brand for the first three months, selecting one in which the fat is not above 2·5 per cent., and then gradually to introduce one of the dried whole milks.

The average caloric value of the dried modified milks after reconstitution with water is from 16 to 18 calories per ounce.

Condensed Milk. Condensed milk is prepared by evaporating cow's milk *in vacuo* down to about one third of its original bulk. It is sold in two forms, sweetened and unsweetened, the sweetened variety having cane sugar added to it. The percentage composition of these milks *before* being reconstituted with water is shown in the following table :

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[1] *Lacidac* (Cow and Gate). This is a dried milk which has been acidified by the addition of one drachm of lactic acid to one quart of milk. It is sold in three strengths corresponding to full cream, half cream, and separated Cow and Gate.

Condensed (Evaporated) Milks.

Brand of Milk.	Protein.	Fat.	Carbohydrate
Sweetened Varieties.			
Nestlé's ("Milkmaid" Brand)	9·4	10·2	53
Diploma	8·4	9·6	54·6
Unsweetened Varieties.			
Nestlé's ("Ideal" Brand)	8·8	9·5	12·2
Libby's	9·6	9·2	11·5
Carnation	8·8	9·2	11·3

The sweetened brand is reconstituted by mixing one part of milk with eight parts of water. This dilution reduces the amount of carbohydrate to the proportion present in breast milk, but the percentage of fat and protein then becomes very low—only just above 1 per cent. Sweetened condensed milk is easily digested, and will often be found useful when dealing with premature babies, or feeble marasmic infants. It tends to give a rapid rise in weight by virtue of its high sugar content, but babies who are fed on it for several months are likely to develop rickets owing to its very low fat content, and their resistance against infections is lowered. It should only be used for a few weeks, until such time as the infant is able to tolerate a richer food, and in the meantime one of the concentrated preparations of vitamin D (*e.g.* Radiostol, three drops daily) should be given. Unfortunately when once an infant has been started on sweetened condensed milk considerable difficulty is likely to be experienced in making a change on to a better balanced food.

Unsweetened condensed or evaporated milk is in quite a different class, and can be used most successfully in infant feeding, indeed it is a pity that the popular use of the term "Condensed milk" refers generally to the sweetened rather than to the unsweetened variety. Care must always be taken to prevent infection of the milk when once the tin has been opened. The following table shows the appropriate dilution of the milk with water and the percentage composition thus obtained. The last column shows the amount of sugar to be added :

	Proportion of		Percentage of			
	Ideal or Libby's Milk.	Water.	Protein.	Fat.	Lactose.	
0–3 months	1 part	3 parts	2·3	2·3	3	Add a level teaspoonful of sugar to every four ounces.
3–6 ,,	1 ,,	2 ,,	3·0	3·1	4	

Occasionally in infants who seem unable to tolerate feeds of the usual size it becomes necessary to give smaller and more concentrated feeds. Evaporated milk is then worth a trial; it should be given in a strength of two parts of the milk to three parts of water, sweetened with the addition of sugar, and the digestion of the protein safeguarded by adding thirty drops of

Foods intended as additions to Milk.

Brand of Food	Protein.	Fat.	Carbo-hydrate.	Remarks
Benger's Food . .	10	1·0	79	Consists of wheat flour and pancreatic extract, which, when made to directions, converts most of the starch into soluble forms.
Mellin's Food . .	10·35	0 16	79·5	A completely malted food, containing no starch. A useful means of adding carbohydrates before six months of age. Has a laxative action.
Savory & Moore .	10·3	1·4	83·2	Wheat flour and malt diastase. When made with milk to directions, most of the starch is converted into dextrins, and the resulting composition is protein 1·9, fat 3·5, carbohydrate 6 6.
Sister Laura's Food .	15·4	1·9	79·9	Much of the starch is converted into dextrins during preparation, leaving only 1·7 per cent. of soluble starch.
Allenbury, No. 3 .	8·8	1·4	84	Wheat flour and malt, containing about 60 per cent. starch.
Chapman's Whole Flour	9·4	2·0	79	Finely ground wheat flour. Contains much unaltered starch.
Farex (Glaxo, Ltd) .	15·0	2·5	71 5	Wheat, oats, maize, bone flour, dried yeast, iron and vitamins A and D. Contains much starch.
Frame Food . .	13·4	1·2	79	Baked flour with added sugar. Contains much unaltered starch.
Neave's Food . .	10·5	1·0	80	Baked flour. Contains much starch.
Nestlé's Milk Food .	14·5	6·0	75	Dried Swiss milk with baked flour and cane sugar. Contains about 18 per cent. of starch.
Ridge's Food . .	12 13	2·7	79·7	Baked flour. Contains much starch.
Robinson's Patent Barley	5·1	0·9	82	Ground pearl barley. Contains much starch.
Robinson's Patent Groats.	11·3	1·6	75	Ground oats from which the husk has been removed. Contains much starch.

lactic acid (B.P. strength) to the pint of the mixture. The replacement of vitamin C in the diet by daily amounts of fresh fruit juice is as essential in this form of infant feeding as in any other.

Foods Intended only as Additions to Milk. Nearly all the foods of this class contain starch, and as a rule should not be given under six months of age. They then become useful not only as a method of introducing starch in an easily digestible form, but also as a means of thickening the feed, and so increasing its nutritive value, without adding to its bulk. A feature of all these foods is their high percentage of carbohydrate, while fat is almost entirely absent, and so they are only suitable as infant foods provided that they are prepared with milk or milk-and-water mixtures.

The opposite table shows the percentage composition of some of these foods. The first four foods contain little or no starch when made according to the manufacturer's directions, and therefore they can be used for infants below six months of age. Their addition to a milk-and-water mixture will not affect the fat or protein content, but will increase the carbohydrate, and should symptoms of carbohydrate indigestion arise they must of course be discontinued. They are not required in the case of an infant who is thriving satisfactorily, but are of great service for the undernourished infant with a small stomach capacity, who can only tolerate small amounts of food without vomiting. They are also a most useful means of thickening a feed in such conditions as aerophagy and rumination.

The foods in this group are used in the strength of one or two tablespoonfuls to the pint of milk or milk-and-water. Their caloric value lies between 100 and 120 calories per ounce of powder.

Hypersensitivity to Cow's Milk. Alarming symptoms have occasionally resulted from hypersensitivity to cow's milk. Tisdall and Erb reported two infants, and Ashby a third, and in each instance the symptoms arose when cow's milk was given for the first time. After one or two mouthfuls the infants screamed and refused to continue the milk feed, and this was followed in a few minutes by acute œdema of the lips, tongue, and face. Drops of milk falling on the skin gave rise to urticarial wheals. The œdema passed off in about an hour, but returned immediately if more milk was given. Other symptoms that have arisen include intense vomiting, fever, the passage of loose stools sometimes containing blood, and collapse.

Treatment consists of desensitising the infant by giving minute doses of milk by mouth—doses that are too small to produce any reaction. Ashby gave only ·01 of a minim of milk three times a day. The amount should be gradually increased day by day until after three or four months a normal amount of milk can be tolerated. While the desensitisation is being carried out, it may be difficult to give the infant a sufficient diet. If the baby is not sensitive to goat's milk or ass's milk, these can be used in place of cow's milk, but generally the infant is susceptible to the milk of these animals as well as to cow's milk. Wet nursing may then be employed, or else a diet must be prepared from cereal foods made with water, with the addition of cod-liver oil.

Goat's Milk [1]

Goat's milk and cow's milk are very similar in composition, and therefore when goat's milk is used for infant feeding it should be diluted in the same way as cow's milk. It has a stronger odour and taste than cow's milk, and although the goat is not susceptible to tuberculosis, so that there is no risk of that particular infection being conveyed by the milk, it may be a means of infection by the micrococcus melitensis unless the milk is boiled.

Ass's Milk

The composition of ass's milk is shown in the following table, the figures being an average of the analyses of several authors:

Percentage Composition of Ass's Milk.

Protein	Fat	Carbohydrate (Lactose).
1·8	1·1	6·0

The protein and sugar values resemble those of breast milk, but the fat is deficient, so that the milk should only be used as a temporary expedient in the feeding of infants, and indeed this will generally be ensured by its high price. Ass's milk is slightly laxative, but this property is lost if the milk is boiled.

WEANING

A mother should aim at feeding her infant from the breast until he is nine months old. The change that then takes place from breast milk to a more mixed diet constitutes weaning, but it should be clearly understood that the weaning process is not

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[1] Caprolac (Cow and Gate) is a preparation of dried goat's milk. When one part of the powder is diluted with eight parts of water the milk has the following composition: protein 3·4 per cent., fat 1·8 per cent., lactose 5·9 per cent.

WEANING TABLE

Up to 6 Months.		6–7 Months.		7–8 Months.		9 Months Onwards.	
6 a.m.	Breast or bottle feed.	6 a.m.	Breast or bottle feed.	6 a.m.	Breast or bottle feed.	8 a.m.	Cereal 8 ozs. Rusk. Egg yolk ; or Fried bread.
10 a.m.	Breast or bottle feed.	10 a.m.	Cow's milk, 6 ozs. Water, 1 oz. Sugar, 1 teaspoonful. Thicken with a heaped teaspoonful of one of the cereals on p. 64.	10 a.m.	Milk, 8 ozs. Sugar, 1 teaspoonful. Thicken with 2 heaped teaspoonfuls of one of the cereals on p. 64. Feed if possible from a spoon.		
2 p.m.	Breast or bottle feed.	1.30 p.m.	Red gravy, chicken broth, vegetable purée (p. 68) or bone and vegetable broth, 1 oz. Followed by Breast or bottle feed.	1.30 p.m.	Broth, 2–3 ozs. ; occasionally thickened with a teaspoonful of mashed potato or sieved vegetable. Followed by Breast or bottle feed.	12.30 p.m.	Broth, 4 ozs., or gravy ; thickened with potato and vegetable purée. Milk pudding, pulp of stewed fruit.
4 p.m.	Fruit juice.	4 p.m.	Rusk with butter or honey. Fruit juice.	4 p.m.	Rusks and fruit juice.	4 p.m.	Rusks, sponge finger, and fruit juice.
6 p.m.	Breast or bottle feed.	6 p.m.	Breast or bottle feed.	6 p.m.	As at 10 a.m., but using a different cereal.	6 p.m.	Cereal, 8 ozs.
10 p.m.	Breast or bottle feed.	10 p.m.	Breast or bottle feed.	10 p m.	Breast or bottle feed. Some healthy infants will forego this bottle feed.	10 p.m.	Milk and water, 6 ozs.; only if the child wakes for it.

a sudden affair. Additions to a breast milk diet should begin at six months, and during the next three months further additions should be made while the number of breast feeds is gradually reduced, so that when the time comes to stop breast feeding the infant has already learned how to digest other foods.

The process of weaning can most easily be explained by taking the case of an infant being fed from the breast every four hours. At six months of age starch should be introduced by replacing the 10 a.m. breast feed by one of the cereal foods such as Farex, Groats, Patent Barley, Allenbury No. 3, etc. These have to be made up with cow's milk, and at first the milk should be diluted. Milk six ounces, water an ounce, with a teaspoonful of sugar and a full teaspoonful of the cereal would be a suitable proportion. As soon as a tooth is through, generally at six or seven months, a rusk or Robb's biscuit with a little butter or honey on it should be added, and may conveniently be given at the ordinary teatime with a drink of orange juice. At 1.30 p.m. an ounce of veal broth or chicken broth or red meat gravy should be given, and the baby should be encouraged to take this from a spoon. A bone-and-vegetable broth [1] is very suitable and has the advantage of preparing the way for sieved vegetables at a later date. Tinned purées [2] of mixed vegetables suitable for infants are now obtainable, and may be given in place of home-made broth; they are more nutritive, easily digested, and much simpler to prepare.

Between seven and eight months the 6 p.m. breast feed should be replaced by a cereal feed as well as the 10 a.m. feed, and at this age there is no need to continue diluting the milk. The feed should be made up to eight ounces, and it is better to give different cereals in the morning and evening. The amount of broth or gravy at midday should be increased to two or three ounces, and two or three times a week it should be thickened by mashing it into two teaspoonfuls of well-boiled potato or sieved green vegetable.

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[1] Break up 1 lb. of veal or beef bones with a little meat left on them, cover with water and simmer for an hour. Then add chopped up vegetable—potato, carrot, turnip, greens—and simmer for another hour. Strain. Allow the fluid to set into a jelly, which will keep for two or three days if boiled up each day. Begin at six months by giving an ounce at midday, and a month or so later give twice the amount, and begin to thicken by occasionally adding two teaspoonfuls of mashed potato or sieved green vegetable.

[2] Libby's Homogenised Foods. Purées of six different combinations can be obtained as follows:—Peas, beans, asparagus tips; pumpkin, string beans, tomatoes; carrots, spinach, peas; whole milk, whole wheat, soya bean flour; prunes, pineapple juice, lemon juice; tomatoes, celery, carrots, chicken livers, barley, onions. Each tin holds 4½ ozs. The food simply needs warming, and then may be fed direct from a spoon. Similar foodstuffs are prepared by Nestlé's.

At nine months the three remaining breast feeds will be given up, and the times of feeding will now approximate to the normal adult meal-times. The 6 a.m. feed will be dispensed with, and the 10 a.m. feed will now be given at 8 a.m. The 1.30 p.m. breast feed will be replaced by the broth and vegetable meal at about 12.30 p.m. The last breast feed at 10 p.m. will be replaced by cow's milk, but it should only be given if the child wakes for it, and it is as well to dilute the milk with equal parts of water as an inducement to the infant to go without it.

Addition of Vitamins[1]

Careful attention must always be paid to the vitamin content of the diet whether the infant is raised on the breast or artificially. Orange juice should be given every day from the third month, and bottle-fed babies should begin even a little earlier, in the second month. At first a teaspoonful should be given daily, the amount being slowly increased until the juice of a whole orange is taken at six months. The juice should be diluted with water and sweetened by the addition of sugar. The time of day when the fruit juice is given is immaterial, but it should be given always at the same time each day, otherwise it may easily be forgotten. Occasionally infants refuse orange juice, and its place may then be taken by tomato juice, or, less effectively, by grape juice.

During the winter months a teaspoonful of cod liver oil should be given twice a day. The oil should be introduced gradually, beginning with half a teaspoonful at a month, and taking a month to reach the full dose, which should then be continued until two years of age. If cod liver oil emulsion is given (50 per cent. oil) the dose must be doubled. This will supply approximately 650 international units of vitamin D daily. Should the oil be badly tolerated, an equivalent amount of vitamin D can be given in the form of ten drops (5 minims) of halibut liver oil, Radiostoleum, Adexolin or a similar concentrate. During the summer months, provided the infant is out of doors morning and afternoon, a supplement of vitamin D to the diet is not necessary.

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[1] As a wartime measure, H.M. Government distribute through the Infant Welfare Centres "Cod Liver Oil Compound," a teaspoonful containing 800 I.U. vitamin D and 3,500 I.U. vitamin A. A teaspoonful daily is sufficient for infants and young children, except those prematurely born who should receive double this amount. The shortage of oranges has led to a similar distribution of black currant purée, and rose hip syrup ; two teaspoonfuls twice daily supply sufficient vitamin C A 25 mgm. tablet of ascorbic acid daily will serve equally well.

Diet between Nine and Twelve Months

The meal times should by now have been adjusted to the customary hours of adult meals. Unless the child sleeps during the morning, fresh fruit juice should be given at 11 a.m.; otherwise it is given at teatime. The thick cereal foods should be given from a cup and spoon, so that the bottle is given up before the end of the first year. The total amount of milk per day at this period should be from twenty to twenty-five ounces. The following table shows the management of the meals for the day :

8–8.30 a.m.	Cereal (eight ounces) e.g. Groats, Patent Barley. Followed by yolk of egg or fried bread, and a rusk spread with butter or honey.
12.30 p.m.	Bone and vegetable broth, or chicken or veal broth, or meat gravy (four ounces), thickened with mashed boiled potato and sieved vegetable. Occasionally yolk of egg, if not given at breakfast. Followed by milk pudding (custard, cornflour, ground rice, sago) two tablespoonfuls, with the pulp of stewed apple or prune. A little cold water to drink at the end of this meal.
4 p.m.	Two rusks with butter and honey, or Robb's biscuit or sponge cake, or a little white bread and butter. Fresh fruit juice may conveniently be given at this meal, or else in the middle of the morning if the child does not sleep then. If fruit juice is given in the morning, a small cup of boiled milk can be given at teatime.
6 p.m.	Cereal (eight ounces) as at breakfast time.
10 p.m.	A feed should only be given if the child wakes for it. It should consist of equal parts of milk and water.

Diet after One Year

After the first birthday the use of a feeding bottle should be definitely dispensed with, and the child should take his fluids from a cup and spoon. The amount of milk per day should not exceed one pint. Jersey milk is too rich in fat for many young children, for whom milk from a mixed herd is preferable. The 10 p.m. feed should be stopped at twelve months if not previously, the last feed consisting of a cup of warm milk given at 6 p.m. With many children this bedtime feed can be stopped with advantage at about eighteen months, the last meal being taken at teatime.

The management of the diet now consists of adding gradually to the variety of foods. At twelve months, a slice of peeled raw apple may be added to the breakfast ; the whole of a softly boiled egg should be given at three breakfasts per week, alternating with fried bread or half a crisply fried rasher of bacon. At about fifteen months the type of breakfast cereal should be changed to one of the more solid varieties such as Force, Cornflakes, Shredded Wheat or Rice Crispies.

After twelve months steamed white fish may be added occasionally to the midday meal, and at fifteen months a tablespoonful of scraped steak, pounded chicken, or minced beef, may be given. Ordinary meats finely cut up should be allowed towards the end of the second year. Steamed puddings should occasionally replace the milk puddings after eighteen months. Stewed fruit (without skins or pips), seedless jam, jellies, junket, and thoroughly ripened mashed banana may be included at any time after the first year.

CHAPTER V

DIGESTIVE DISORDERS IN INFANCY

Introduction. In order to deal successfully with the digestive disorders of infancy a full history of the previous management and feeding must first of all be obtained. It is not sufficient merely to know the name of the last food that has been tried. The history should include such details as the number of feeds in the day, and their amount and strength ; the time taken over the feeds ; whether additions of sugar, cream, cod-liver oil, and so on are being given ; the number of stools passed in the day, and their colour and consistency. Without a knowledge of these facts the source of the trouble is likely to remain obscure.

Most of the digestive troubles at this age can be traced to the infant receiving either too little or too much food, or else to one of the main components of the diet, namely, the protein, fat, or sugar, being badly tolerated. Of these various causes, underfeeding is the most common.

UNDERFEEDING

The management of underfeeding in breast-fed infants has already been considered (p. 52). Underfeeding in bottle-fed babies may come about for various reasons—for instance, the amount of food at each meal may be insufficient, or the spacing of the meals may be too far apart. Insistence upon four-hourly feeding, especially in the early months, often leads to trouble of this sort, and as a rule it is more satisfactory to feed an infant every three hours (six feeds a day) for the first three months, and then to change over to four-hourly feeding (five feeds a day). Another not uncommon cause of underfeeding is that the hole in the teat of the bottle is too small. What happens then is that the infant sucks for perhaps half or three-quarters of an hour without emptying the bottle, and eventually gives up exhausted. In bottles that have a rubber valve at one end to let in air, the valve may be too stiff to work properly, and so the infant has to suck against an increasing vacuum. Then sometimes the quality of the food is too poor. This applies but seldom to breast milk unless it happens that the mother is taking a very deficient diet, but it is not at all uncommon to find that an infant receiving a milk-and-water mixture is having the milk too dilute, or that a dried or condensed brand of milk is being prepared at too weak a strength.

Symptoms. The symptoms vary according to the degree of underfeeding. Often there is a history that the infant is restless and cries after a feed, or he may lie quietly for an hour or so and then wake up and cry until the next feed is given. The weight may rise too slowly, or may remain stationary, and if the underfeeding continues for some weeks the baby may become miserably thin. Vomiting does not as a rule occur, although if a great deal of air is swallowed at meal times some milk may be regurgitated with each eructation. Constipation is usual, and is sometimes so severe that two days or more may go without a stool being passed, or, on the other hand, the baby may pass three or four green stools a day, but these amount to little more than a bile staining of the napkins—the so-called "hunger-stools."

Treatment. The underlying cause must first be determined. Should the infant be unable to digest a milk-and-water mixture at a strength appropriate to the age (see p. 58), a change should be made to a dried milk instead of diluting the milk still further, since this would simply aggravate the underfeeding. The dried milks, it will be remembered, are for the most part reconstituted by allowing one drachm of milk powder to one ounce of water ; or if unsweetened condensed milk is used, one part of milk should be diluted with three parts of water. Then the correct amount of food for the whole twenty-four hours must also be estimated by allowing two and a half ounces of fluid for every pound of body-weight, and this amount must be divided into the number of feeds which it is decided to give. The feeding bottle should also be inspected to make sure that the valve and teat are working properly.

After the diet has been correctly adjusted, a small dose of chloral should be given before each feed for a week or two. To an infant of three months, $\frac{1}{2}$ gr. of chloral can be given in a teaspoonful of sweetened water. The purpose of the drug is to break the infant's habit of restlessness and crying after the feeds, getting him instead to settle down peacefully when the meal is over.

OVERFEEDING

Overfeeding is not so common as underfeeding. It may come about either because the feed is too rich, or more usually because too great a quantity is being given. Breast milk is seldom too rich, nor is a mixture of cow's milk and water, although Jersey milk, owing to its high content of fat, may disagree when ordinary cow's milk suits perfectly well More often a too rich feed results from insufficient water being used for the reconstitution of dried milk. It is never wise to allow more than one drachm of milk powder to be mixed with an ounce of water, and even then a "full-cream" brand of dried milk may, in virtue of its high content of fat, prove too strong. In that case the substitution of a "humanised" or half-cream brand will often be all that is required.

The quantity may be excessive for various reasons, but it is particularly likely to come about when the feeding is irregular. One is sometimes told that an infant is allowed to suck at the bottle until he is satisfied, regardless of the amount he is taking, and this almost always leads to considerable overfeeding.

Symptoms. Overfeeding is very likely to be overlooked at first, because there is to begin with a rapid gain in weight and the infant seems to be doing very well. There may be a little regurgitation of milk at the end of the feed, but this so commonly happens in quite healthy babies that no notice is taken of it. The stools at first also appear normal, although they are large and increased in number. If overfeeding continues the digestion eventually becomes overtaxed, and characteristic symptoms arise. The regurgitation of milk increases until most of the feed may be vomited, and at the same time the stools become more frequent, loose, and slightly offensive, and may contain small yellow curds. The abdomen becomes distended and tympanitic owing to excessive fermentation in the intestine, frequent attacks of colic occur in which the infant screams and draws up his knees on to his abdomen, and much flatus may be passed from the bowel. The weight which was rising quickly, now checks or may fall, and unless treatment is promptly adopted, the infant may in the end become considerably wasted. The following table shows a comparison of the symptoms of underfeeding and overfeeding.

Symptoms Indicating

UNDERFEEDING	OVERFEEDING
Failure to gain weight.	Rapid gain of weight at first, then failure to gain and eventually loss of weight.
Vomiting generally absent.	Vomiting—at first small amounts after feeds, later on larger vomits.
Flatulence mostly gastric, causing colic and screaming.	Flatulence from intestinal fermentation, leading to abdominal distension with colic and screaming.
Stools generally constipated and small. Small green "hunger stools" may be passed.	Stools large, tend to be frequent. Later they become loose, curdy, and may be offensive.

In a later section the symptoms that arise from indigestion of protein, fat, and carbohydrate, are considered as separate entities, clear-cut examples being met with when the diet contains one of these elements in excess, but it must be pointed out here that a general level of overfeeding may lead to symptoms suggestive of one or other of these substances being particularly badly tolerated, or alternatively there may be a combination of the symptoms met with under each separate heading.

Treatment. Treatment is always a protracted affair, for it is not merely necessary to reduce the amount of food, but the digestion has also to be nursed back into a healthy condition. A teaspoonful of castor oil should be given at the outset in order to empty the bowels of any undigested residue of food When deciding the composition of the new diet, it is wise to begin with a weaker food than might at first seem to be required. Cow's milk should be diluted to a half-and-half strength ; or a full cream dried milk should be replaced by a humanised or half-cream brand (such as half-cream Cow and Gate), and for the first week or two this should be prepared at a weaker strength than usual by allowing one drachm of milk powder to every ounce and a half, instead of every ounce, of water. As a rule it is better to weaken the strength of the feed than to reduce its bulk, for infants who have been overfed will become dissatisfied and irritable if the size of their meals is reduced, although they will tolerate a reduction in the strength. The reduction in diet can sometimes be more easily carried out by giving half an ounce of boiled water at the beginning of the feeds.

As in the treatment of underfeeding, a small dose of chloral should be given at the beginning of each feed for a week or two, to help the infant to settle down when the meal is over.

PROTEIN INDIGESTION

Indigestion of protein is not so common as was at one time believed, for it is now realised that many of the symptoms which were originally attributed to the casein curd are the outcome of indigestion of fat or carbohydrate. The increasing use of dried milk, in which the casein is rendered more digestible, has also helped to make protein indigestion less common.

Symptoms. These are most likely to be met with in infants who are reared on fresh cow's milk, the caseinogen of which is converted in the stomach into large tough casein curds, where they may remain for some time, giving rise to a mucous catarrh. The most prominent symptom consists of vomiting of the curds with a good deal of mucus, while the efforts of the stomach to drive on the curds may account for sharp attacks of colic and screaming. The stools are usually constipated, slightly alkaline, and occasionally contain a few brown leathery-looking curds. With these symptoms there is a failure to gain weight, or actually some loss of weight.

Treatment. In severe cases it may be necessary to remove the casein entirely from the diet, but more often it is sufficient to dilute the milk or to modify the casein by one of the methods about to be described, or alternatively to substitute a dried milk. In addition, the catarrhal state of the stomach should be dealt with by repeated gastric lavages (for method see footnote), otherwise vomiting is likely to continue. Gastric lavage is more easily carried out in infants than in older people, and with much less disturbance to the patient. It should be employed daily for the first two or three days, and then be continued every other day until the vomiting stops or the lavage ceases to bring back any curd or mucus.

There are various ways of assisting the digestion of casein.

(1) *Citration.* The addition of sodium citrate to milk prevents the formation of insoluble calcium caseinate (ordinary curd), soluble sodium caseinate being formed instead. The curd that forms in citrated milk is fine and light. The maximum effect is obtained by allowing 1½ gr. of sodium citrate to every ounce of whole milk, and there is no advantage in exceeding this dose. To take an example, if an infant is having four ounce feeds of equal parts of milk and water each feed will contain two ounces of whole milk, and therefore the dose of sodium citrate would be 3 gr. The amount of sodium citrate should be prescribed in a drachm of water, one drachm being added to each feed. If larger doses of citrate are used there is a risk of causing œdema or of starting a troublesome diarrhœa.

(2) *Acidification.* The value of acidifying cow's milk lies in

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Gastric Lavage. The apparatus consists of a glass funnel, such as the barrel of a glass aural syringe, and 3 feet of soft rubber œsophageal tubing. The largest size of tubing that can easily be passed should be used, and the tube should have a large eye at its lower end. The tube is dipped in glycerin and then passed quickly over the tongue and down the œsophagus for about 10 to 12 inches. A solution of bicarbonate of soda (a drachm to the pint of water) is the best fluid to use. The tube should be pinched near the infant's mouth while the funnel and tube are filled with fluid and air bubbles emptied. Then three or four ounces of the solution are allowed to run into the stomach. Just before the funnel empties, the tube is pinched again and the funnel inverted below the level of the child so as to siphon off the gastric contents, including pieces of curd and mucus. This should be repeated until the returning fluid is clean. The amount drawn off must measure the same as the amount run in.

the fact that it is richer in " buffer substances ", chiefly phosphates and caseinates, than is breast milk. A buffer solution has the property of combining with moderate amounts of acids or alkalies without undergoing any change in reaction. When cow's milk reaches the stomach a certain amount of the gastric hydrochloric acid is first of all soaked up by the buffer substances, and not until this has taken place is there any acid available for the processes of digestion. This can be overcome by artificially acidifying or souring the milk sufficiently to satisfy the buffer substances. The acidification can be carried out bacterially by adding a culture of bacillus bulgaricus to the milk and incubating for twelve to twenty-four hours, but such a process is too elaborate for ordinary household use. The same result can be achieved more simply by adding either lactic acid or dilute hydrochloric acid to the milk (for preparation see footnote).

In spite of its slightly sour taste, infants usually take an acid milk very well. It is not only of value when there is difficulty in digesting protein, but can also be used in wasting conditions, particularly after gastro-enteritis, for it does not require to be so diluted as ordinary fresh milk, in fact if the milk is first of all skimmed of some of its cream and then acidified, it can as a rule be given undiluted.

(3) *Peptonisation.* Predigestion of the protein by peptonisation [1] affords another means of overcoming protein indigestion. According to the method of preparation described in the footnote, complete peptonisation is reached in thirty to forty minutes. At first the milk should be fully peptonised, and then as the digestion improves the time of peptonisation should be gradually reduced, until finally a return is made to ordinary milk. Peptonised milk should only be used as a temporary measure, for its prolonged use is likely to leave the infant under-nourished and frail. As a rule

Lactic Acid Milk. Boil one pint of fresh milk ; when cold, add drop by drop forty-five minims of lactic acid (B.P. strength, 75 per cent.), stirring all the time. This should take about fifteen minutes. Towards the end a very fine curd forms which will pass through the ordinary teat. The milk should be warmed and sweetened before being given to the infant.

Lacidac (Cow and Gate) is a dried lactic acid milk, made in three strengths, corresponding to the ordinary Cow and Gate milk foods, namely, full-cream, half-cream, and skimmed.

Hydrochloric acid milk is prepared in exactly the same way as lactic acid milk by adding forty-five drops of acid hydrochlor. dil. (B.P. strength).

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[1] *Peptonised Milk.* Add to a pint of milk and water a cupful of Fairchild's peptogenic powder, or a peptonising tablet (Parke, Davis & Co.). Stand the milk in a vessel of hot water for from twenty to forty minutes, according to the degree of peptonisation required, and then raise the milk to the boil. Whole milk does not peptonise easily ; the proportion of milk and water will depend upon the state of the infant's digestion.

it is possible gradually to slide the infant on to a more normal diet after two or three weeks.

(4) *Whey.*[1] Another method is to remove the casein entirely from the milk, as is done in the preparation of whey (for method of preparation, see p. 60). Whey consists of the water, lactose, lactalbumin, and mineral salts of milk, with not more than a third of the fat, and is too weak a food to nourish an infant for very long, in fact it should not be used alone for more than about a week. Both the fat and the sugar can be increased so as to bring the values up to those of breast milk by adding half a teaspoonful of cream and a teaspoonful of sugar to every four ounces of whey, but this is more successful in theory than in practice owing to the fact that infants with curd indigestion are so liable to tolerate fat badly, and in practice a better method of strengthening the whey is to thicken it with one of the cereal foods, such as Savory and Moore's or Mellin's or Benger's, by adding a tablespoonful of one of them to half a pint of whey. In deciding which cereal to use, it should be remembered that Mellin's tends to have a laxative effect, while Savory and Moore's has a slightly costive effect. As the condition of the infant improves, the whey should be gradually replaced by ordinary milk, until finally the infant is taking a milk-and-water mixture thickened with one of the cereals.

Sherry-whey [2] is sometimes of advantage over ordinary rennet-whey by virtue of the stimulant action of its alcohol, and it may be used when the symptoms of curd-indigestion have been present for some time and there is considerable emaciation, but after a week or so attempts should be made to replace it by a more nourishing diet such as the whey and cereal-food mixture just described.

FAT INDIGESTION

Of the various elements in cow's milk, the fat is the one most likely to be badly tolerated, and symptoms of fat indigestion are particularly likely to arise in young infants who are brought up from birth on a full-cream brand of dried milk. It is for this reason that the author prefers, during the first three months of life, to use one of the partially-skimmed or half-cream dried milks, in which the fat has been skimmed down to 2·5 per cent. (see table, pp. 63, 64). Fat indigestion is seldom met with in infants who are receiving milk-and-water mixtures, unless it should happen that Jersey milk is being used, or the diluted milk is being enriched by the addition of cream.

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[1] *Secway* (Trufood Ltd.). This is a preparation of dried whey, the powder consisting of lactalbumin, 13 per cent.; lactose, 76 per cent.; mineral salts, 9 per cent.; and having a caloric value of 106 C. per ounce.

[2] *Sherry-whey.* Add two ounces of cooking-sherry to half a pint of milk which has been heated to just below boiling. Take the milk to the boil, stand for a few minutes, and strain through muslin. An ounce of sherry-whey made according to the above directions contains the equivalent in alcohol of twenty-five drops of brandy (Still), so that if sherry-whey is being given, alcohol in other forms should not be prescribed.

Symptoms. The evidence of indigestion of fat is mostly derived from the character of the stools. At first the fat is excreted in the form of soaps, and the stools are bulky, formed, and pale in colour, and are often so firm that the infant has considerable trouble and pain in passing them. At this stage, the urine as it lies in the napkin often develops a strongly ammoniacal odour, and the buttocks may become reddened and even ulcerated. Later, free fatty acids are passed in the stools, which then become loose, strongly acid, and offensive, and their passage is likely to cause much pain and screaming. Vomiting is not usually a prominent symptom, at any rate at first, but later on sour-smelling material may be returned. Nutrition is soon interfered with, and the weight falls.

Treatment. This consists of cutting down the amount of fat in the diet. An initial dose of castor oil should be given to clear the intestine of irritating soaps and fatty acids. In mild cases it may be sufficient simply to change from a full-cream to a half-cream dried milk, or if fresh cow's milk is being given it should be skimmed of its fat. Considerable care will be needed in re-introducing fat into the diet, and for some weeks it should not rise above 2 per cent. In the case of infants who are being fed on dilutions of fresh cow's milk, additional cream must be entirely avoided, but after a few weeks a small teaspoonful of cod-liver oil emulsion may be given once a day.

When the symptoms have been more severe, a completely skimmed dried milk, such as Cow and Gate special separated milk, may be used for a week or two before introducing a half-cream brand. The sweetened variety of condensed milk, or Horlick's Malted Milk, both of which contain only a low content of fat, will also be found useful for these more severe cases, and may be given for two or three weeks, but if they are to be continued longer, cod-liver oil should be added to the diet.

Buttermilk, which is that part of milk remaining after the churning of butter, contains less than 1 per cent. of fat, and so can also be used in the treatment of fat indigestion. Commercial buttermilk is prepared from milk which has been soured by lactic acid bacilli, and therefore needs to be sterilised by boiling (stirring all the while to prevent curdling) before being used as an infant food. It is an acid milk, with a composition of protein 3 per cent., sugar 3·5 per cent., and fat 0·25 to 1·5 per cent. It should be sweetened by adding a teaspoonful of sugar to every three ounces. Eledon (p. 63) is a preparation of dried buttermilk, and skimmed Lacidac has a similar composition.

Red and excoriated buttocks should be treated with some soothing application such as zinc oxide and castor oil in equal parts, and the napkin should be lined with a layer of cotton wool. If the skin over the buttocks has not broken, a simple dusting powder, such as equal parts of zinc oxide and starch, should be freely used. A more detailed description of napkin rashes will be found on p. 631.

CARBOHYDRATE INDIGESTION

Indigestion of carbohydrate is not so common as indigestion of fat, and when it occurs it will generally be found that the carbohydrate has been added in the form of starch—perhaps as barley water, but more often as one of the proprietary foods containing starch. Badly balanced foods which have an excess of sugar, such as the sweetened form of condensed milk, are also likely to cause symptoms of carbohydrate indigestion, although many infants can tolerate sugar in the form of mono- and di-saccharides up to 3 or 4 per cent. above the percentage present in breast milk. Carbohydrates play an important part in assisting the tissues to retain water, and so long as the digestion remains healthy, a high carbohydrate diet causes a big gain in weight due to the amount of water retained. Although infants fed in this way quickly put on weight, their tissues are flabby, and in the event of an attack of diarrhœa, to which they are more than normally prone, the drop in weight is rapid and profound.

Symptoms. The symptoms arise from excessive bacterial fermentation of the carbohydrate in the intestine. Distension of the abdomen, due to the accumulation of gas in the intestine, is a common symptom, and in long-standing cases this produces a wide separation of the recti muscles, so that when the infant attempts to sit up a ventral bulge appears between the tightened muscles. Attacks of colic with screaming and drawing up of the legs are frequent, and an excessive amount of flatus is passed. The stools become loose and green, and may actually appear frothy from the amount of gas contained in them ; they are acid in reaction, but are not so offensive as the stools of fat indigestion. Within a day or two the buttocks become inflamed and the skin may excoriate. Vomiting is not an early symptom, but appears later as the breakdown of digestion becomes more complete. The gain in weight, which may at first have been rapid, soon becomes arrested, and a severe degree of wasting may eventually come about.

Treatment. Treatment should begin with a teaspoonful dose of castor oil. The amount of carbohydrate in the diet must then be reduced, and if starch has been given it must be entirely withheld. A simple mixture of milk and water will be found most satisfactory. It will be necessary to sweeten the mixture, and for this purpose the most suitable sugar is a mixture of dextrins and maltose, sold under the name Dextri-Maltose or Daltose, since this is less fermentable than cane-sugar, glucose, or lactose. Enough should be added to bring the carbohydrate content up to about 6 per cent., which is a little less than the amount present in breast milk. If equal parts of milk and water have been ordered, a teaspoonful of sugar should be added to every four ounces of the mixture ; or if the milk and water is in the proportion of 2 to 1 a teaspoonful of sugar should be added to every five ounces.

Protein milk,[1] which incidentally is an acidified milk, is also successful in these cases, as its proportions of sugar, and to a less extent the fat, have been reduced. Prolonged feeding on a low sugar diet is likely to lead to constipation, and then the amount of sugar should be gradually increased until the bowels are working normally.

Although drugs are only of secondary importance, a combination of chloral and castor oil in small doses is useful at the beginning of treatment to check the diarrhœa and to rest the infant. After the initial purgative dose of castor oil, the following mixture may be given three times a day :—

R Chloralis Hydratis gr. ss–i
Olei Ricini m. v.
Mucilaginis Acaciæ m. xv.
Aquam ad ʒi.

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[1] Protein milk (Finkelstein formula) can be obtained in a dried form from Mead, Johnson & Co. It is reconstituted by adding one drachm of powder to one and a half ounces of water—a dilution which differs from most dried milks—and when made up in this strength has the following compositions : protein, 3·1 per cent. ; fat, 2·25 per cent. ; sugar, 2·0 per cent. ; lactic acid, 0·18 per cent.

VOMITING

When an infant is brought on account of vomiting, one must first be sure that it is something more than the mere regurgitation, or " possetting ", of a teaspoonful or so of milk at the end of the feed, which is of such common occurrence as to have no particular significance.

The next step must be to exclude causes arising outside the gastro-intestinal tract. The onset of acute infections such as bronchitis, pneumonia, otitis media, and pyelitis, is often indicated by vomiting ; at times the eruption of a tooth is preceded by vomiting which may persist for several days until the tooth has finally penetrated the gum ; meningitis, hydrocephalus, and even intracranial neoplasms, are other possible causes.

As a general rule when vomiting is a symptom of indigestion it is accompanied by diarrhœa, or at least by the passage of undigested motions. Vomiting in association with constipation is at all times a combination of symptoms worthy of special notice. Occasionally these two symptoms date from birth, and may then be due to some form of congenital obstruction in the alimentary tract such as stenosis of the œsophagus or obliteration of the duodenum or small intestine, conditions which are more fully considered elsewhere. The onset of forcible vomiting associated with constipation within a few weeks of birth will suggest hypertrophic pyloric stenosis.

Two special types of vomiting remain to be considered ; one is associated with aerophagy, the other with rumination.

Aerophagy

In this condition the infant swallows excessive amounts of air, generally during a meal, and the big eructations that follow are accompanied by the regurgitation of much of the feed. It has already been pointed out that infants normally swallow a certain amount of air at their meals. Excessive air-swallowing generally originates in some simple technical fault such as the hole in the teat of the bottle being either too large or too small, or the feeds being given at too long intervals. There is no doubt that the use of a dummy may sometimes start off a habit of air swallowing; as too may the habit of thumb-sucking.

Although aerophagia generally begins from such simple causes as those enumerated, if it continues the infant may get into a habit of swallowing air, and can be heard between his mealtimes gulping down large amounts of air with evident enjoyment. Noisy eructations follow, and are accompanied by the regurgitation of milk. Most of the previous meal may be lost in this way, and unless the habit is checked the weight soon becomes stationary and eventually a severe degree of malnutrition may be reached.

Treatment. The technique of the feeding must be carefully overhauled, the small points to which attention has just been drawn being corrected. The air-swallowing at mealtimes can sometimes be checked by giving smaller and more frequent feeds, and by giving a little boiled water at the beginning of the meal to check the infant from sucking too vigorously. If these measures do not suffice, the feeds should be thickened by the addition of half to one tablespoonful of Benger's or Savory and Moore's Food.

A sedative and carminative mixture after meals is often helpful, a suitable prescription being :—

℞ Chloralis Hydratis gr. ss.
Spiritus Ammoniæ Aromaticus m. iiss.
Tincturæ Cardomomi Compositi m. iii.
Glycerini m. v.
Aquam Menthæ Piperatæ ad ℥i.

Rumination (Merycismus)

This consists of the regurgitation of milk from the stomach into the mouth, where it is chewed and tasted and then swallowed again.

Rumination is almost entirely confined to bottle-fed babies, and rarely occurs before four months of age. The baby has a good appetite and takes his feeds well, but a few minutes afterwards he can be seen to twist and wriggle his body until his mouth gapes and fills with milk. The whole performance gives obvious pleasure to the infant, and the process is continually repeated. With each regurgitation some of the milk is spilled from the mouth, until almost the whole feed may be returned. The loss of nourishment may be great enough to give rise to considerable emaciation, and indeed unless effective treatment can be devised the condition may prove fatal. According to Grulee (quoted by Thomson) the mortality is as high as 25 per cent.

The cause of this curious condition is uncertain. In the early stages the infants are generally big and strong, and it is likely that the habit is formed from the possetting of a little milk, which so commonly happens with infants who receive a slightly too large feed. In others, the regurgitation of small amounts of milk after aerophagia has forerun this distressing habit. When once the trick has been learned, it quickly becomes a habit which may be difficult to eradicate.

Unless the process has actually been observed, there is often little in the history to direct attention to the presence of rumination. The usual story is that the infant has done well until about four or six months of age, but since then has vomited its feeds, has become constipated, and has failed to thrive. Further questioning may show that the vomiting is not forcible, but after a feed the clothes are found constantly soiled by vomit—as soon as one bib is changed, the next one becomes wet. The diagnosis cannot be considered proven until the habit has been actually seen. It is of little use to stand in front of the baby as these infants seldom perform to an audience, but by observing the child from behind the cot, or by watching him through a screen, the whole process may be witnessed. The retching movements, the sight of milk in the mouth, the chewing and swallowing, will then leave no doubt of the diagnosis.

Treatment. Treatment to be successful must be thorough. The habit has to be broken, and it is a wise move to begin by putting the baby under the care of a trained nurse. The most useful method is to thicken the feed so as to make regurgitation very difficult. The milk may be thickened with Groats or Arrowroot or Farina to such a consistency that it will scarcely run from a tilted spoon. The habit interferes with the sleep that should normally follow a meal, and therefore a sedative, such as 1 gr. of chloral, should be given with each feed. Another method is to prevent the infant from opening his mouth, without which the act cannot be performed, by putting on a strong linen bonnet with side straps which fasten under the chin by a buckle. The straps can then be pulled together just sufficiently to keep the mouth closed. Although this method is often employed it has the danger that if the infant attempts to ruminate he may possibly inhale some of the milk, and for this reason the author prefers to rely upon thickening the feeds. Treatment must be continued for at least a month to ensure the habit being completely broken.

FLATULENCE AND COLIC

These two symptoms are commonly associated together and are a source of much discomfort in infancy. An excess of carbohydrate in the diet, especially starch, is a common cause, owing to the gaseous distension of the intestine and the irritation caused by the strongly acid contents. Excessive air-swallowing, often associated with underfeeding, is another common cause. Constipation, with hard fæcal masses in the intestine, is also likely to lead to colicky attacks, which may continue until the bowel has been emptied. It must be remembered that a sudden onset of recurring colicky pain in a previously healthy infant may be due to an acute intussusception, but the passage of a little blood and mucus from the bowel, together with a palpable tumour in the abdomen, should prevent this condition from being overlooked.

A typical attack of intestinal colic begins suddenly, the infant screaming and drawing up his thighs on to the abdomen, which becomes tense and rigid. Each attack lasts for two or three minutes, and if the pain is unusually severe the infant may become pale, and afterwards may appear exhausted. The attack passes off gradually, but may recur at frequent intervals, and so prevent the infant from getting any rest or sleep. Vomiting may occur during the attack, and in nervous infants the risk of a convulsion is always present. Occasionally the stools are constipated, but more usually they are loose, green, and contain small yellow fat curds. Flatulence and colic of any severity are generally associated with a failure to thrive, or an actual loss of weight.

Treatment. Screaming attacks may arise from other reasons than intestinal colic, and when an infant is brought on account of screaming, other causes, such for example as acute otitis media or acute pyelitis, must be excluded before assuming that the intestine is at fault.

Immediate relief of pain is best given by the local application of heat. Warm fomentations or poultices should be applied to the abdomen, and an enema of two or three ounces of warm water will sometimes soothe the infant, and, by assisting the passage of flatus or hard fæcal masses, may at the same time remove the exciting cause. When the colic is so severe that the infant seems in danger of collapse, a warm bath or a mustard bath[1] is often the best restorative.

To prevent further attacks the diet must be corrected, any excess of sugar being reduced, and starch entirely withheld;

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[1] *Mustard Bath.* An ounce of mustard should be mixed to a smooth paste with cold water, and then stirred into a gallon of warm water at 100° F. The infant should be held in the bath for five or ten minutes, or until the nurse's arms begin to tingle. The child should then be dried by dabbing, not by rubbing, and be wrapped in a warm blanket.

the amount of the diet must, of course, be enquired into to prevent underfeeding, and it is as well to watch the infant being fed, which may bring to light some technical error.

A sedative mixture such as that given on p. 85, to which three drops of tincture of belladonna may be added, will prove beneficial. When the stools are loose and undigested small doses of opium, such as Dover's Powder (gr. $\frac{1}{8}$ at three months or gr. $\frac{1}{4}$ at six months, four times a day), may be given with advantage for a few days, but should be used only as a temporary measure until the alteration of diet has had time to bring about an improvement.

CONSTIPATION

The importance of training an infant to open his bowels at definite times of the day cannot be exaggerated, for many of the most resistant cases of habitual constipation in older children owe their origin to lack of proper training in infancy. From the first week or so after birth the baby should be held out over a small chamber after each meal, being taken off as soon as the bladder or rectum has been emptied. By assiduous effort in this way, the infant soon learns to associate the feel of the chamber with the passage of urine and fæces, and good habits will be learned. Training on these lines also does something to lessen the chance of irritation and soreness of the buttocks, which is usually produced by prolonged contact of the skin against a soiled napkin.

The most common cause of constipation in infancy is underfeeding, with the result that very little residue reaches the lower bowel, and the stools, which are small, are passed perhaps only every second or third day. It is useless to treat these infants with purgatives, correction of the constipation can only come about by increasing the daily intake of food. Occasionally too small an amount of water may be being given, although the nutritive value of the diet is perfect. The stools are then larger, but are very hard and dry, and the infant is likely to be restless because of thirst. The trouble is easily corrected by giving extra water.

Then apart from the actual amount of food, constipation may result from the components of the diet being badly balanced. A high ratio of protein or of fat tends at first to produce large hard constipated stools, while a low proportion of sugar has the same effect. The addition of more sugar, up to about 8 or 10 per cent., may be all that is required. Some sugars are more laxative than others, cane-sugar in the form of ordinary brown demerara sugar being particularly useful in this respect. The addition of Mellin's or Benger's Food to the diet, a teaspoonful to every six ounces, will serve the same purpose. Although fruit juice is given every day solely for its anti-scorbutic value, the slightly laxative effect is at times helpful ; but a mother should never be encouraged to give fruit juice for this purpose, for then she may withhold it unless her infant is constipated, and so allow him to run a risk of scurvy.

Congenital stricture of the anus is an occasional cause of constipation. It is more common in female infants, and there is usually a history of much straining and screaming before a small hard stool is passed. During the passage of the stool the mucosa may be torn, and the stool will then be streaked with a little bright blood. The diagnosis can only be made by doing a rectal examination, and it may here be pointed out that even in the youngest infant digital examination of the rectum can safely be performed, provided that the finger is well greased and its insertion through the anus carried out slowly. Normally the finger slips in after a few seconds without much difficulty, but when there is anal stenosis the opening is very small and tight, and it may be some minutes before the tip of the finger passes through the tightly gripping rim of the anus. Not only does a digital examination enable the diagnosis to be made, but, by stretching the sphincter, the condition may be permanently relieved.

Very rarely a congenital stricture may occur at the upper end of the rectum. This was so in an infant who was brought when a month old to King's College Hospital, with a history of severe constipation since birth, and failure to gain weight. Examination showed a distended abdomen, and peristaltic movements of the intestine were clearly visible. By digital examination in the rectum a narrow stricture could be felt just within reach of the finger at the recto-sigmoid junction.

Constipation is a very common feature of mental defect, both in infancy and in later childhood. It is largely a matter of habit, the children being either incapable of appreciating the ordinary demands of nature, or too lazy to respond to them. Training them in regular habits calls for much perseverance on the part of the parents, but it is essential if habitual constipation is to be prevented. Although all grades and varieties of mentally defective infants are likely to be constipated, it is particularly a feature of untreated cretinism.

There remain two other conditions in infancy in which constipation is a prominent feature, namely hypertrophic pyloric stenosis and Hirschsprung's disease or idiopathic dilatation of the colon.

Constipation is likely to give rise to other symptoms. Attacks of colic and screaming, particularly when a stool is being passed, have already been mentioned. A failure to thrive is sometimes another prominent symptom, and that it depends on the constipation is proved by the steady gain in weight when the bowels have been made to work with regularity. A furred tongue, unpleasant breath, coldness and blueness of the extremities, may also be met with in infancy, although they are a more common accompaniment of constipation in older children. There may be nervous symptoms such as restlessness at night and peevishness, which at times may culminate in convulsions. This was so in an infant aged nine weeks, who had been reared on a full-cream dried milk, and who had had repeated convulsions for eight hours. A rectal lavage brought away several hard fæcal pellets, whereupon the fits ceased.

The severe and repeated straining that accompanies constipation is a common cause of hernia, either at the umbilicus or in the inguinal canal. Piles are quite exceptional in infancy, but when they occur they are almost always associated with constipation.

Treatment. The various points in the diet to which attention must be directed have already been mentioned. In the majority of infants, correction of the diet will put an end to the constipation, and resort to the use of drugs should only be made after the feeding has been carefully overhauled.

When using drugs it is better to employ them daily in laxative doses than to wait until the infant is constipated and then to give purgative doses. For this reason castor oil, which is so popular a remedy in the home, is a bad drug for chronic constipation, indeed small doses actually promote constipation while larger doses have an initial purgative effect followed by constipation. For young infants magnesia is the most suitable laxative. A teaspoonful of Phillip's Milk of Magnesia may be given with the first feed in the morning ; it should be mixed with the last ounce of the milk and be given either from a spoon or the bottle. If grey powder (Hydrargyrum cum Creta) is used, a laxative dose, such as ¼ gr. or ½ gr. twice a day, would be suitable, but this preparation finds its greatest use when dealing with constipation in a feverish infant, the dose being then increased to one or two grains in a single administration. Liquid paraffin acts as a pure lubricant, and is useful when the stools are hard, and only passed after much straining ; the dose should be such that the oil just does not separate in oily drops from the stool ; a small teaspoonful once or twice a day is usually sufficient. Paraffin emulsions, such as Petrolagar, Agarol, Nujol, also contain a laxative principle, and are more certain in their action than plain paraffin. Paraffin should not be used when there is already a good deal of mucus in the stool.

For infants who are more severely constipated, syrup of senna (m. 20 to 30 at night) will often be found a most convenient preparation, and owing to its sweetness it is readily taken. When constipation is associated with mental defect, small doses of thyroid should be given, and this not merely in cases of cretinism. To an infant of six months, thyroideum siccum, gr. $\frac{1}{5}$ once a day will as a rule be a sufficient dose.

Lastly mention must be made of suppositories and enemata. These are used much too frequently. They should only be employed now and again when oral methods have failed, for not only does their regular use make the anal sphincter patulous, but they do not encourage the rectum to respond to its normal stimuli, and in fact infants are likely to remain constipated so long as reliance is placed solely upon them. As a suppository, a small paring of plain soap is the least harmful and it can generally be relied upon to produce a stool. Glycerin suppositories are also often employed, but they are likely to cause some local irritation. When the rectum is filled with hard scybalous masses a warm enema of plain water or soap and water should be given, three or four ounces being run in gently at a temperature of 100° F. It is often useful to precede this by an enema of an ounce of warm olive oil about half an hour beforehand in order to soften the fæcal mass.

DIARRHŒA

Introduction. Infantile diarrhœa is a common condition which can conveniently be divided into three main groups :

(1) Dietetic diarrhœa.
(2) Infective diarrhœa.
(3) Parenteral diarrhœa.

In each group vomiting is almost always associated with the diarrhœa, and the symptomatic title of " Diarrhœa and Vomiting " is often employed as though it connoted a disease. The term

"Gastro-enteritis" is also used, indicating that inflammation of the stomach and intestines is the cause of the symptoms, although it must be admitted that it is usually impossible to demonstrate such inflammation at post-mortem examination.

Diarrhœa has its greatest incidence during the first year of life, and the mortality is relatively higher then than at any other age. In spite of the high incidence in the first year, severe diarrhœa and vomiting is not at all common among breast-fed infants. At The Hospital for Sick Children, Great Ormond Street, in a consecutive series of 100 infants below nine months of age who were admitted on account of diarrhœa, only three were being fed entirely at the breast, and a further six were receiving breast milk supplemented with cow's milk. Infants reared on the sweetened brand of condensed milk show the greatest liability to diarrhœa.

Dietetic Diarrhœa

Errors in diet account for the majority of cases of infantile diarrhœa at the present day, at any rate in this country. The fault may lie in various directions, for instance the total amount of food per day may be too great, so that the digestive capacity is overtaxed and sooner or later breaks down. An excess of fat soon leads to the passage of free fatty acids in the stools, and they become loose, curdled, and sour-smelling. Parents are sometimes so anxious to get their infants on, that they lavishly add extracts and emulsions of all kinds to the diet. While in moderation these extras are generally harmless, their injudicious use is very likely to provoke an attack of diarrhœa. But it is to excess of carbohydrate, usually in the form of starch, that dietetic diarrhœa is most often due, particularly when the starch is introduced at too early an age, before the sixth month. The loose, green, acid, and frothy stools which then result are a clear indication of the excessive amount of fermentation going on in the intestine.

The incidence of dietetic diarrhœa is distributed more or less evenly throughout the year. As a rule the illness may be classed as mild, and in this respect, as also in the lack of any seasonal incidence, dietetic diarrhœa differs very definitely from infective diarrhœa. The mild nature of dietetic diarrhœa is, however, not to be relied upon, indeed if the error in the diet is not quickly corrected the diarrhœa may rapidly exhaust the infant, or may initiate a gradual loss of weight which eventually leads to severe and fatal marasmus.

Infective Diarrhœa

This term implies that the diarrhœa is due to an infection of the gastro-intestinal tract, although bacteriological investigations have failed to show any one particular organism as the cause. Cultures made from the stools, and from scrapings of the intestinal mucosa taken after death, have grown various organisms such as streptococci, B. proteus, B. enteridis sporogenes, B. lactis aerogenes, B. paratyphosus, dysentery bacilli, Morgan's bacillus and the Sonne bacillus, while more often than not only the normal flora of the intestine have been present. It is also possible that the chemical changes consequent upon bacteriological decomposition of milk play a part. The condition is very much less likely to affect breast-fed infants, unless they should happen to be brought into contact with other cases and so become infected.

The incidence of infective diarrhœa varies at different seasons of the year, being highest in the late summer months, at a time when the mean atmospheric temperature is at its highest. Until a few years ago annual epidemics of infantile diarrhœa, generally of a severe type and carrying a high mortality rate, swept over the country during the months of July, August and September, and although during the last few years these epidemics have declined remarkably, indeed almost to vanishing point, the incidence of infective diarrhœa still remains highest in the third quarter of the year.

When " summer diarrhœa " was prevalent its incidence varied from one year to another, depending on whether the summer was a hot one or not—the summers of 1898, 1899 and 1911 were exceptionally hot, and in these years the number of infants dying from diarrhœa rose to a higher level than at any time in the preceding thirty years, and such figures have fortunately never been approached since. Precisely how such hot weather influences the incidence of infantile diarrhœa is uncertain, but the increased amount of dust and number of flies makes it more likely that milk and food will be contaminated, while the rapid multiplication of bacteria in warm weather increases their virulence.

During the last ten years or so the incidence of infantile diarrhœa in the summer has fallen to figures almost as low as obtain in the winter. The accompanying chart, which is made from the returns of the Registrar-General, gives the mortality of infantile diarrhœa for each quarter of the year from 1913 to 1932, and shows that the decline in mortality has occurred chiefly in the third quarter, and to a less extent in the fourth quarter, while the figures for the first half of the year have remained more or less constant. That the number of deaths from diarrhœa in the first half of the year has not appreciably altered is explained by the fact that they are largely due to the dietetic and parenteral types, and it is of interest to note that the summer figures have dropped to, but not below, the winter figures, because of course the dietetic and parenteral diarrhœas continue to occur evenly throughout the year.

Several reasons can be advanced to account for the very satisfactory reduction in summer diarrhœa. The encouragement of breast feeding, the very improved hygienic methods in the collection and distribution of milk, the growing custom of sterilising fresh milk by boiling or pasteurising, and the increased use of dried milk, have all played their part. The replacement of horse traffic by motor transport, which has incidentally had the effect of diminishing the amount of dust and the number of flies, may also be a factor.

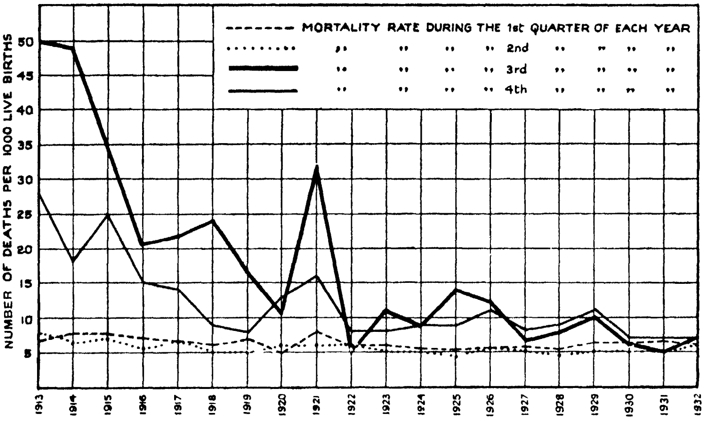

FIG. 7. Chart showing the quarterly mortality rate from enteritis and diarrhœa in infants under two years of age in England and Wales for the years 1913 to 1932.

Infective diarrhœa is as a rule a much more severe illness than the dietetic variety, and indeed may be so brisk as to overwhelm an infant within a few hours.

Parenteral Diarrhœa

This term is used for those cases, by no means uncommon, in which the diarrhœa is secondary to some condition outside the gastro-intestinal tract. The fact is that any acute infection in a young infant is liable to give rise to vomiting and diarrhœa, indeed these symptoms may be looked upon as taking the place of headache and malaise in older people. Thus they may be associated with acute otitis media and mastoid disease, or with acute infections of the respiratory tract, and they are common symptoms at the onset of acute pyelitis, and may also occur in meningococcal meningitis. Many infants experience a fleeting attack of diarrhœa with the eruption of each tooth.

Parenteral diarrhœa is often a sharp illness, and unless the possibility of infection in some other part of the body is always borne in mind, it is very easy to overlook the secondary nature of the diarrhœa. This mistake can only be avoided by taking a careful history and by making a thorough examination, including in all cases the throat, the ears, and the urine. The diarrhœa has, of course, to be treated, but unless the primary illness is also dealt with the treatment of the diarrhœa is likely to prove ineffective.

Pathological changes in infantile diarrhœa. The post-mortem findings in infants who have died from diarrhœa are remarkably few, and often there is nothing abnormal to be made out in the stomach or intestines. A general congestion of the bowel is occasionally present, and is seen to best advantage if the intestines are bunched together, when the prevailing colour of the whole mass is pinker than normal. This must, of course, be distinguished from post-mortem staining which affects the more dependent parts of the bowel. When during life there has been mucus and blood in the stools—such cases are often designated as ileo-colitis—there may be one or several small circular ulcers, little larger than a pin's head, in the lower end of the small intestine and colon. The mesenteric glands are not noticeably enlarged. A striking and almost constant finding is fatty degeneration of the liver, due probably in large measure to the passage to the liver of toxic substances derived from the bowel. In this connection, Boyd [1] has found that the portal blood of fatal cases of infantile diarrhœa contains a toxic substance allied to histamine, which if injected into animals causes a rapid collapse. The degree of destruction of the liver is probably the most important factor influencing the prognosis, although it is very difficult to assess; certainly when the course is rapidly downhill and no treatment seems to make any appreciable difference, the amount of liver damage is always extreme. When the diarrhœa has lasted for a few days before death, fatty degeneration may also be found in the heart muscle and in the kidney. Collapse of the bases of the lungs is a common finding, and a terminal broncho-pneumonia, often unsuspected during life, may also be present.

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[1] Boyd, *Arch. Internal. Med.*, 1923, **31**, 297.

Clinical Picture. In the mild cases, due most often to errors in the diet, there is often a preceding history of digestive difficulties. The onset may be acute with vomiting or diarrhœa as the initial symptom, or there may be a gradual increase in the number of stools. These are loose, half a dozen or more being passed in the day, and are at first green, slightly offensive, and often contain small fatty curds and sometimes a good deal of mucus. The temperature is raised two or three degrees, the anterior fontanelle is depressed, and the infant is miserable and continually wailing. The loss of fluid in the stools causes the weight to drop and restricts the output of urine.

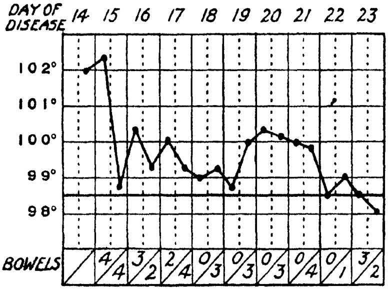

FIG. 8. Temperature chart of an infant aged three months, who recovered from an attack of diotetic diarrhœa.

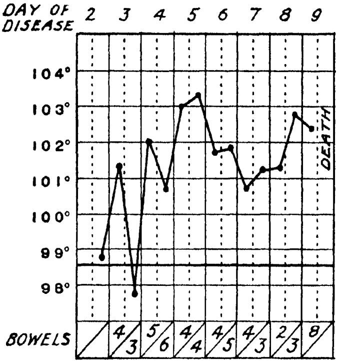

FIG. 9. Temperature chart of an infant aged four months, who died from infective diarrhœa.

In the more severe cases, which may be either dietetic or infective in origin, the onset is usually brisk, and there may be an initial convulsion. Vomiting is an early symptom, and even water may be returned so that it is impossible to get the infant to retain any fluid given by mouth. At other times vomiting may be slight or even absent, although the condition of the infant is none the less serious. Diarrhœa is urgent, and the stools, which are at first green and curdled, soon become watery and odourless. The rapid loss of fluid quickly plunges the infant into a condition of severe dehydration; he becomes pale and restless, the temperature is raised to 103° or 104° F. or even higher, the anterior fontanelle loses its normal resilience and pulsation and becomes very depressed, the eyes are sunken, and the skin loses its elasticity, so that when it is pinched up it remains wrinkled instead of springing back to its former position. The tongue becomes dry and red, and the papillæ often appear unusually prominent; the abdomen becomes flabby and sunken, and peristaltic movements of the small intestine may be visible. There is seldom any abdominal tenderness. The pulse becomes rapid, mounting to 160 or more per minute, and the heart sounds are soft and tic-tac in character. Albuminuria is usually present, and is associated with a diminished flow of urine, indeed in fatal cases there may be complete anuria for twenty-four hours before death.

To such an unhappy picture the symptoms of toxæmia are later added. The face then becomes livid and pinched, the restlessness disappears and the infant lies apathetic on his back, the eyes stare so that mucopus often collects on the cornea, the respirations become very shallow, so that all that may be seen or heard of them is an occasional sigh. The pulse may be uncountable and the heart action becomes irregular; the extremities appear cyanosed, and the temperature may either continue high until the end or may fall to subnormal. Death may seem gradually to steal over the child, or may be heralded by one or more convulsions.

It not uncommonly happens that although the immediate treatment is successful and the infant rallies, diarrhœa of a milder order continues for several weeks, the weight fails to rise, and gradually the infant passes into a condition of marasmus from which recovery, if made at all, is a long and difficult process.

In the most severe grade of all, the so-called "Cholera Infantum," the whole course of the illness is accelerated. The onset is sudden with violent purging, the stools quickly have the appearance of rice-water, while the symptoms of intoxication—staring eyes, sighing respirations, rapid irregular heart action, and perhaps hyperpyrexia—quickly supervene. Fortunately such cases are becoming much less common with the disappearance of summer diarrhœa.

Mention has already been made of the particular type of infantile diarrhœa spoken of as ileo-colitis. The infection in these cases falls mainly on the ileum and colon. There may be no vomiting, but diarrhœa is severe, and the stools contain much mucus and a little blood. The abdomen is likely to be distended and slightly tender, but in other respects these cases resemble those already described.

Complications. Examination of the mouth may show the presence of "thrush", due to infection by the fungus *oidium* *albicans.* This appears as small white firmly attached patches on the tongue and inside the cheeks, and may sometimes initiate the attack of diarrhœa. At all events the diarrhœa is not likely to cease until the thrush has been disposed of, which is easily accomplished by cleansing the mouth several times a day with glycerin and borax, or painting with 1 per cent. aqueous solution of gentian violet.

Otitis media has already been mentioned as a cause of parenteral diarrhœa, but it may also develop as a complication. When this happens there is likely to be an increase of the vomiting and diarrhœa, the temperature shows a further rise, and the infant may indicate the source of his pain by screaming and pulling at his ears. In addition there may be some stiffness of the neck and a positive Kernig's sign, the meningeal symptoms being due to irritation of the meninges (meningismus), for as soon as the ear is dealt with they pass off and the diarrhœa lessens. Broncho-pneumonia is also a serious complication, and is one which is very likely to be overlooked, particularly in toxic infants who have lain moribund for some days before death. There may be neither rise of temperature nor increase of respiration rate to make one suspicious, and the presence of consolidation is revealed unexpectedly at post-mortem examination.

Skin complications are generally a warning of a bad prognosis. Small abscesses may form in the subcutaneous tissues, particularly over the occipital region or on the back. In prolonged cases purpura is not uncommon, appearing as petechial hæmorrhages on the trunk and over the lower abdomen. Occasionally, when death is preceded for a day or so by a state of collapse and a subnormal temperature, patches of the subcutaneous fat over the shoulders and limbs become firm, feeling as though solidified (sclerema). Little can be done for this beyond the application of warmth and general stimulant measures, as in most cases this curious change is a terminal event.

Thrombosis of veins is an occasional complication, the large veins or the cerebral sinuses being affected. On two occasions the author has found at autopsy thrombosis of the renal vein, the kidney on the affected side being swollen and purple, but in neither case had hæmaturia occurred, indicating that the thrombosis had taken place only a short while before death. Thrombosis of the cerebral sinuses is also a late event. It may be indicated by convulsions, or there may be a combination of irritative and paralytic phenomena. This was so in the case of an infant five months old, who was admitted to hospital suffering from infective diarrhœa. A day or so later the left side of the face became paralysed, the pupils were of pin point size, and the limbs were stiff and making continual fighting movements. A diagnosis of meningeal hæmorrhage due to a sinus thrombosis was made, and a cisternal puncture was performed ; the cerebrospinal fluid was under increased pressure and pink in colour from the uniform admixture of blood in it. Following the puncture the movements of the limbs ceased, only to return a few hours later when the child died. Convulsions may occur at the onset of an attack of diarrhœa, and are then as a rule not of much significance, but their occurrence during the course of the illness is always serious and may, as in the case just quoted, indicate some grave intracranial disturbance.

Treatment. Although certain guiding principles can be laid down for the management of infantile diarrhœa, each case requires separate consideration ; it is seldom possible to plan a course of action to cover more than a few hours because the condition of the infant may undergo such rapid alterations, and the doctor must be prepared to adjust his treatment accordingly. Frequent observations of the infant's progress are essential.

The three principles that underlie the treatment are (1) to rid the bowel of any food or toxic material that may be accounting for the condition. (2) To replace the fluid that has been lost by the diarrhœa. (3) To rest the stomach and intestines for at least a day or two before making a gradual return to a suitable diet. In any but the mildest cases skilled nursing is required, and a trained nurse should be employed. A close watch must be kept on the temperature, which may either become hyperpyrexial, calling for a quick sponging, or, in the face of collapse, may fall several degrees below normal.

At the outset it is generally wise to empty the bowel by giving a purgative dose of castor oil, allowing half a teaspoonful for every six months of age, although if the diarrhœa has already been severe enough to deplete the infant of fluid, or if the child appears collapsed, the preliminary dose of castor oil will have to be omitted as it would simply tax the infant's strength unnecessarily.

Washing out the stomach and bowel is also a valuable means of helping to get rid of toxic material. Gastric lavage (for method, see p. 78) is particularly useful when vomiting is persistent, especially if much mucus is being brought up. It is a much less disturbing performance in infancy than in older subjects, but even so it may be too exhausting for a feeble infant, and stimulants should always be at hand. Irrigation of the colon may be of great benefit in cases of ileo-colitis, the bowel being gently washed out with six or eight ounces of warm saline at 100° F.

Every effort should be made to replace the fluid which has been lost in the stools. This can be attempted by giving repeated sips by mouth of water, glucose water (5 per cent.), or albumen water,[1] reckoning to get in an ounce or more every hour. If repeated vomiting makes this impossible, normal saline containing 5 per cent. of glucose may be given per rectum by means of a continuous drip enema, but the bowel must be washed out first of all otherwise the fluid is not likely to be retained. Unfortunately the intensity of the diarrhœa is likely to prevent fluid being given by the rectum, and then if the symptoms of dehydration are severe, as would be indicated by the depressed fontanelle, sunken eyes, and inelastic skin, saline must be given by some other route, either subcutaneously, intravenously or intraperitoneally.

The subcutaneous injection of normal saline,[2] to which 2½ per cent. of glucose water has been added, is the most convenient method. The injection is usually made into the loose tissues below the axillæ, from three to six ounces being run in slowly on each side. The amount that is given will depend on the size of the infant and the speed with which the saline is absorbed. As a rule absorption is rapid, the whole injection being taken up within an hour. Subcutaneous salines may be repeated once or twice a day until the infant is able to retain fluid by mouth; consecutive injections should be made into fresh areas, using the outer side of the thighs or the flanks as well as the axillæ.

When the danger from loss of fluid seems more urgent, the injection of saline may be given directly into the circulation. The continuous intravenous drip method [3] is the most satisfactory, having the advantage that the infusion can be continued if necessary for several days at a time, and meanwhile the amount of fluid by mouth need be only enough to keep the tongue and throat moist. The injection is usually made into the median basilic vein at the elbow or the internal saphenous vein in front of the internal malleolus. In infants and young children it is always necessary to expose the vein by making a small incision and raising it on an aneurysm needle. Use has also been made of the external jugular vein and the superior longitudinal sinus, but the risk of air embolism in the former, and of causing a sinus thrombosis in the latter, makes either of these routes undesirable. If normal saline is used, after a day or two the infant is likely to show some slight œdema owing to the defective elimination of salt through the kidney, and it is therefore better to use half-normal saline, to which 5 per cent. of glucose should be added. Hartmann's solution (an isotonic solution containing sodium lactate, sodium chloride, potassium chloride, and calcium chloride) may also be employed with good effect.

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[1] *To prepare Albumen Water.* Cut the white of an egg in several directions with sharp scissors. Shake it with half a pint of cold water, and strain.

[2] *To give Subcutaneous Salines.* The apparatus consists of a funnel (the barrel of a glass aural syringe serves very well) connected by rubber tubing to a Y-shaped glass connection, to which two hollow needles are attached by short rubber tubes. The apparatus, and the fluid for injection, must be sterile. The saline should be heated to about 130° F. before being given, as it cools considerably by the time it reaches the tissues. Before the needles are inserted through the skin the whole apparatus must be filled with the saline in order to exclude any air. The fluid is then allowed to run in slowly by gravity, the funnel being held about 12 inches above the child.

[3] *Continuous Intravenous Saline.* The apparatus consists of a container for the saline fixed above the infant. This is connected by rubber tubing to a glass dropper and air-trap, which in turn is connected by rubber tubing to a needle tied into a vein at the elbow, the arm being splinted. Just before its attachment

The direct injection of saline into the peritoneal cavity has its advocates. The advantages of this route are that a greater quantity of saline can be given more speedily than by the other methods, and absorption is also quicker than from the subcutaneous tissues. To carry out the injection the bladder must first be emptied and then the abdominal wall is picked up off the intestines and the needle is inserted slantwise in the middle line. Five to ten ounces of saline are then allowed to run by gravity into the peritoneal space. The decision to employ this route should not be left until the infant is already moribund, as the saline may then not be absorbed at all. Although the danger of pricking the intestine is said to be very slight the method is not one which appeals to the author, owing to the risk of injury and the danger of peritoneal shock to an already exhausted infant.

When the child is judged to be too ill to recover with saline injections, an improvement can sometimes be brought about by the transfusion of a small amount of whole citrated blood. A preliminary red-cell count should be made, for if the degree of dehydration has been severe the red cells may be so concentrated

to the needle the tubing should be arranged to pass over a hot-water bottle to warm the saline. The technique is facilitated by using special Bateman needles (Allen & Hanbury), which consist of two hollow needles, one fitting inside the other. The larger needle is tied into the vein, while the smaller one is attached to the apparatus and can then be passed through the larger needle to the lumen of the vein. The rate of flow of the saline is regulated by a clip so that from six to eight drops of saline are allowed to run in per minute, which is equivalent to one pint in twenty-four hours.

that the count is well above the normal, and in that case serum would be more valuable than whole blood.

Mention must also be made here of the use of anti-dysenteric serum for those infants with acute infective ileo-colitis from whose stools dysentery organisms have been isolated. An intramuscular injection of 10 c.c. should be given daily.

Diet. At the beginning of treatment a preliminary period of starvation has a beneficial effect by resting the bowel. The period for which food is withheld will vary ; in the mildest cases twelve hours may be long enough, in severe cases the infant may go as long as forty-eight hours without food. Meanwhile, although food is withheld, it is, of course, essential that fluid should be given either by mouth, by rectum, or by infusion.

The introduction of food must be gradual, and it is well at first to give small feeds frequently, such as at two-hourly intervals. After an attack of diarrhœa the most difficult element in the diet is usually the fat, and therefore a food should be chosen in which the amount of fat is low. Fresh milk, even when diluted, is seldom well tolerated. A skimmed dried milk (such as skimmed Cow and Gate) can often be used with success, but it is so weak a food that it should not be used for more than a week or so before a half-cream or humanised brand is introduced. When the illness has been very severe a start may be made with plain whey, or sherry-whey, giving from one to three ounces every two hours according to the age. As the infant improves, generally after three or four days, the diet should be slowly worked up to a more nourishing level by thickening the whey with a little Savory and Moore's Food and by gradually replacing the whey by milk. Owing to its small amount of fat and relatively high content of carbohydrate Horlick's Malted Milk may be tolerated when other foods prove unsuitable, and for the same reasons the sweetened form of condensed milk may also be used with success ; but because of their low fat content these foods should only be used for two or three weeks, or alternatively should be enriched after a few weeks by the addition of a small amount of cod-liver oil emulsion. Lactic acid milk has been highly recommended in the treatment of diarrhœal conditions, but it has seemed more useful when dealing with the chronic wasting which may follow acute gastro-enteritis, than actually within a few days of an attack.

Recently success has been claimed to follow the use of raw apple in diarrhœal conditions in young children. In the writer's hands it has proved of value in both infants and young children when the acute diarrhœa has passed but the stools have remained loose and offensive, the effect being to render them formed and to lose their offensiveness. It is also effective in dysentery. More details of the apple diet are given on p. 217.

Drugs. The value of a purgative dose of castor oil at the outset of treatment has already been mentioned. Later on, small doses of castor oil, such as five minims thrice daily, are well worth giving for their constipating effect. The oil may be prescribed as follows :—

℞ Olei Ricini m. v.
Mucilaginis Acaciæ m. xv.
Aquam ad ʒi.

When vomiting continues for more than a day or two bismuth may prove of benefit, but sufficiently large doses must be given. It may be usefully combined with aromatic chalk powder in the following prescription :—

℞ Bismuthi Carbonatis gr. x.
Sodii Bicarbonatis gr. ii.
Pulveris Cretæ Aromatici gr. v.
Mucilaginis m. xv.
Aquam Chloroformi ad ʒi.

Drugs with an " intestinal antiseptic " action are as a rule useless in the treatment of acute diarrhœa, but when the stools remain persistently undigested and offensive small doses of calomel (gr. ⅛ ter die) or salol (gr. ½ to 1) have sometimes seemed of benefit.

Opium is undoubtedly a most valuable drug in dealing with infantile diarrhœa, but mention of it has been delayed until now because its use entails a careful selection of cases, and the effect of the drug must be closely watched. Opium should not be used when the tongue has become very dry, nor when the symptoms of toxæmia—apathy, shallow respirations, rapid irregular heart action—have replaced those of dehydration, for then the infant is likely to respond excessively to very small doses, lapsing into a coma which may prove fatal. On the other hand small doses of opium are indicated when the diarrhœa is severe in proportion to the vomiting, when the stools are green and tending to be offensive, and in cases of ileo-colitis. Pulv. ipecac. co., gr. ⅛ for every three months of age, is a useful preparation which may be combined in a powder with bismuth carbonate. A liquid preparation is often taken more readily, in which case tinct. opii (m. ⅛ for every three months of age up to a year) may be prescribed. In order to prevent the chance of overdosage with opium, those in charge should be warned never to wake the infant for the medicine, and to omit it if he is drowsy.

Stimulant treatment is called for when the signs of collapse, such as a low temperature, cold extremities, and a feeble pulse appear. A warm bath, or better still a mustard bath, is often the best means of rallying the infant, and it can be repeated once or twice a day as the clinical condition demands. Brandy is also a drug not to be despised ; it should be given every three or four hours in doses of 5 to 10 drops up to three months of age and 15 to 30 drops from then up to twelve months of age. It will often be retained when plain water is vomited. It is probable that the merit of sherry-whey, when the first severity of the diarrhœa has passed, is due in large measure to its alcoholic content. When the need of stimulation appears more urgent, a hypodermic injection of strychnine may temporarily tide the infant over. A suitable dose would be m. ¼ of the liquor strychninæ B.P. up to three months of age, and m. ½ up to twelve months.

INFANTILE WASTING (MARASMUS : ATHREPSIA)

Profound wasting in infancy, even though it may be given such special titles as marasmus or athrepsia (Parrot), does not constitute a disease, but is a symptom dependent upon some underlying cause, for which a careful search must always be made. Even so, there will remain a number of cases in which the cause is quite obscure, and it is with these that we shall here concern ourselves. But mention must first be made of two causes which may be easily overlooked. The first of these is congenital heart disease. When, as may happen in a young infant, there is neither cyanosis nor distressed breathing to direct one's attention to the heart, only a most careful examination will prevent the condition from being missed. Should the infant be crying during examination the bruit over the heart may be almost inaudible, and it is therefore a good rule to listen while the infant is being given a feed so as to ensure a quiet and thorough examination. The second condition is congenital syphilis. As a rule there are other signs to indicate the diagnosis, such as snuffles, rashes, or enlargement of the spleen and liver, but this is not always so. The importance of recognising syphilis as a possible cause lies in the fact that the response to anti-syphilitic treatment is usually good.

Other conditions in infancy in which wasting may be a prominent symptom include hypertrophic stenosis of the pylorus, post-basic meningitis, and an undiscovered empyema or pyelitis. Tuberculosis, although by no means a common cause, may sometimes account for a fatal infantile marasmus, and yet signs of tuberculous infection during life may have been entirely lacking. This was so in an infant who had never thrived, and who at the age of seven months weighed less than 6 lbs. Beyond being miserably thin he showed no other physical signs until a day before death, when a few scattered crepitations were to be heard over both lungs. Autopsy revealed caseous glands in the mediastinum, and both lungs were thickly sown with miliary tubercles.

Of the dietetic causes of wasting, persistent underfeeding is the most common. Most of the nutritional failures in breast-fed infants fall under this heading, and underfeeding in bottle-fed babies is also very common. A gross instance of this was a baby whose diet for several weeks had consisted of four-hourly feeds of half a teaspoonful of Nestlé's Condensed Milk in two and a half ounces of water, which works out at a percentage composition of protein 0·3, fat 0·3, and sugar 1·2 --small wonder that the infant was wretchedly emaciated. Profound wasting may also be the ultimate outcome of an acute attack of gastro-enteritis.

In recent years a great deal of investigation has been made into the biochemical aspects of marasmus. There is no evidence that the infant makes an uneconomical use of the three principal components of food, namely protein, fat, and carbohydrate, when once these have been absorbed from the intestine ; in other words, infantile marasmus is not due to any peculiar vice of metabolism. It also seems that the absorption of foodstuffs from the intestine is carried out satisfactorily ; the fault lies in a failure of the digestive capacity.

One of the most important effects of wasting is a progressive loss of subcutaneous fat, the function of which is to act as an insulating layer against the loss of body-heat, and as a result an undue loss of body-heat takes place which can only be countered by an excessive production of heat. Unless the infant is capable of digesting a sufficiently large amount of food to satisfy the increased demand for heat, the body produces the required amount by burning up any spare tissue. The amount of heat production, measured as basal metabolism, is raised above the normal until the actual weight has dropped to about 35 per cent. below the expected weight. When that degree of wasting has been reached the spare tissues have all been used up, and the essential tissues such as the muscles and viscera are then sacrificed, and as these dwindle so does the production of heat begin to fail, and the temperature falls below normal. The infant's store of glycogen—never relatively as large in a child as in an adult—is rapidly burnt up, so that the fasting blood sugar is invariably low and may be as much as 10 per cent. below the normal value.

The great difficulty in treatment consists in getting these infants to digest sufficient food to counteract their excessive heat loss and at the same time to allow of a gain in weight. Too often the attempt to give a diet of sufficiently high caloric value leads to a breakdown of the digestion, which necessitates cutting down the diet and so increases the degree of wasting.

The post-mortem examination seldom shows anything of note beyond a severe general atrophy.

Clinical Picture. Few sights can be more pitiful than a severely wasted infant. The face becomes pinched and grey, the cheeks are hollowed, the fontanelle is depressed, and the thin inelastic skin sags between the bony points of the skeleton. The limbs appear as sticks round which an ill-fitting skin has been loosely wrapped, and the abdominal wall is often so thin that the slow peristaltic movements of the intestine are easily made out. The temperature, in the absence of complications, may be three or four degrees below normal. Death may gradually supervene from increasing weakness and exhaustion, but sometimes comes suddenly even when the wasting may have been stationary or slowly improving. When recovery is made, it is complete, so that some months later the children appear perfectly healthy.

Complications. Two complications which may appear in any severely wasted infant are œdema and purpura. The œdema, which pits slightly on pressure, appears at first on the back of the hands and the dorsum of the feet, but may later involve the limbs or face. There is no reason to think that the œdema is due to any loss of renal function, for the urine is as a rule free of albumen ; it is to be attributed to changes in the permeability of the vessel walls resulting from their poor nutrition. Purpura appears most commonly over the lower abdomen and back, and has always a grave significance. Tetany may also occur, and may be detected either by carpo-pedal spasms or by an increased irritability of the facial nerve.

Other complications such as thrush, otitis media, subcutaneous abscesses—particularly on those parts exposed to pressure such as the occiput, scapulæ, and buttocks—and a terminal broncho-pneumonia, have been mentioned already as complications of diarrhœa, but they may also add to the risks of a marasmic infant.

Treatment. One of the most important points is the maintenance of warmth, in order to minimise the loss of body-heat. The temperature of the room should be kept at between 65° F. and 70° F., and this is best done by a coal fire, which will ensure a free circulation of air. Draughts must be carefully excluded, the cot should be surrounded with screens, and warm bottles should be placed beneath the blankets. The most gentle handling is necessary, and the baby should be disturbed as little as possible, although occasional changes of position will help to prevent skin infections and pressure sores. The clothing should be as for a premature infant, the baby being wrapped in cotton wool over which a loose woollen jacket can be worn ; the limbs must be wrapped up as well as the trunk, the head should be covered with a flannel bonnet, and the ordinary napkin should be replaced by a pad of absorbent wool, which can be easily changed. Instead of the usual daily bath, the infant should be oiled over with olive oil every other day. The services of a skilled nurse are invaluable.

The most difficult aspect of treatment is the diet. Too often the history is that the wasting began as soon as the infant was taken from the breast. When the mother's milk has entirely failed, the possibility of securing the services of a wet-nurse should never be lost sight of, for that may mark the turning point of the illness. Whatever food is given, small feeds at frequent intervals—every two or two and half hours—are indicated.

It has already been pointed out that the difficulty in feeding these infants lies in getting them to take sufficient nourishment without overtaxing their digestion. The amount of food should be calculated according to the expected weight rather than the actual weight (in contrast with the feeding of healthy babies). This may be differently expressed by saying that while a healthy baby requires a diet which will furnish 45 to 50 calories per lb. of body-weight, a marasmic infant will need at least 70 to 80 calories per lb. The increased caloric requirement will generally have to be given in the form of sugar, which is as a rule well tolerated up to as much as 12 or 15 per cent. Bearing in mind that the marasmic infant is perfectly capable of metabolising his foodstuffs, including sugar, the use of insulin in treatment, which some have recommended, is difficult to justify ; nor, in the author's hands, has it seemed beneficial. Although fat has a higher caloric value than sugar, it is likely to lead to diarrhœa if it is present in amounts above 2 per cent., and even that amount may not be digested.

A half-cream or a skimmed dried milk with dextri-maltose added in the proportion of one teaspoonful to every two ounces of the reconstituted milk is sometimes successful; or the sweetened form of condensed milk (reconstituted with water in a strength of 1 in 8) may be used for a few weeks. If fresh milk is used, it will need diluting with half the amount of water, and it should then be thickened with Benger's, Mellin's, or Savory and Moore's Food, allowing two tablespoonfuls of the cereal for every pint of the milk and water mixture. Of the three foods named above, Benger's has seemed to the author the most satisfactory.

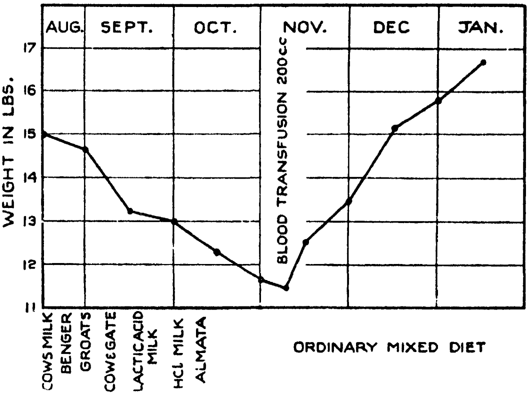

FIG. 10. Chart showing improvement after blood transfusion (200 c.c.) in a boy aged eight months, who was admitted for diarrhœa which progressed to marasmus.

Lactic acid milk has been highly recommended, and with much justification, for it often succeeds when other methods of feeding have failed. One of its advantages is that it can generally be given as whole undiluted milk, but when doing so it is a wise precaution to skim the top cream off the milk before the acid is added, for it is unlikely that the full allowance of fat will be tolerated. The method of preparing lactic acid milk is described on p. 79. It will of course need sweetening, and for this purpose dextrimaltose should be used, allowing up to two tablespoonfuls to the pint of milk. This will raise the carbohydrate value to 12 per cent.

The most valuable guide to the infant's progress is his weight, and this should be recorded every other day. Provided that the digestion remains satisfactory, the caloric value of the diet should be steadily pushed up until at last the weight begins to advance. At this point vitamins A and D, in the form of Radiostoleum or Adexolin, should be added to the diet, allowing 5 drops daily. This should be changed to a teaspoonful of cod-liver oil emulsion thrice daily after two or three weeks, since this is not only a vehicle for vitamins A and D, but is also a useful means of re-introducing fat into the diet. When the stools have become formed, and without waiting for a rise in weight, a teaspoonful of fresh fruit juice should be given each day.

Drugs play but a small part in the treatment of marasmus. Thyroid is sometimes beneficial, although its mode of action is not clear. The dose should be gr. $\frac{1}{20}$ per lb. of body weight per day. Possibly it assists by accelerating the circulation, which is often sluggish and feeble—a suggestion which receives some support from the fact that wasted infants are said to be benefited by being kept in atmospheres containing increased oxygen or increased carbon dioxide.[1]

When the infant seems to be entirely at a standstill, help is sometimes to be derived from small transfusions (two or three ounces) of whole blood. The injection should be made into a vein at the elbow, or into the internal saphenous vein at the ankle. The superior longitudinal sinus at the anterior fontanelle has sometimes been used, but the risks of producing thrombosis of the sinus or of penetrating through the sinus outweigh any advantages of this route. Intraperitoneal injections of citrated blood have also been recommended, but the method does not commend itself to the author. Although the peritoneum is capable of absorbing blood, this is not to be relied upon, as was shown in the case of a marasmic infant six weeks old, who received 70 c.c. of whole citrated blood into her peritoneum. Death occurred four days later, and at autopsy all the blood lay unclotted in the pouch of Douglas.

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[1] McCrea and Raper, *Quarterly Journal of Medicine*, 1929, vol. 22.

CHAPTER VI

HYPERTROPHIC STENOSIS OF THE PYLORUS

In this condition there is obstruction to the passage of food from the stomach, accompanied by great hypertrophy of the circular muscle fibres of the pylorus. The disease is strictly one of infancy, and is almost always confined to the first three months of life.

Etiology. *Age.* Although the condition has been called congenital stenosis of the pylorus, it is very unusual for the symptoms to date strictly from birth ; as a rule the infant thrives satisfactorily for two or three weeks and then the characteristic symptoms appear. Of 129 cases observed by the author at The Hospital for Sick Children, Great Ormond Street, in 69 the symptoms began during the third and fourth weeks, only 5 cases beginning during the first week. The incidence falls rapidly during the second month, and it is very unusual for the onset to be delayed into the third month.

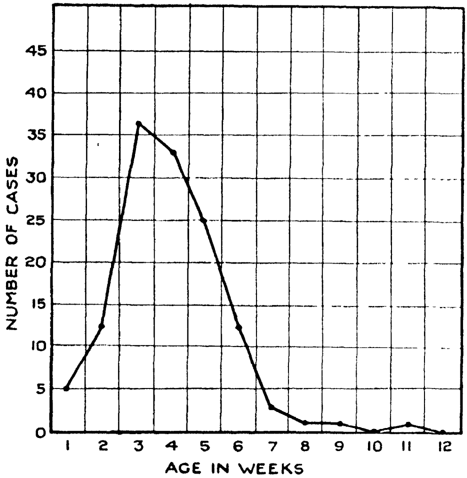

Fig. 11. Chart showing the age at onset of 129 cases of hypertrophic pyloric stenosis.

Sex. There is a remarkable preponderance in males, roughly 6 male infants being affected for every 1 female. Of the 129 cases referred to, 85 per cent. were male infants. Occasionally more than one member of a family has been affected, and the condition has been recorded in both of twins.

Place-in-family. As remarkable as the incidence in males is the peculiar likelihood of the first-born of a family to be affected. This was so in 63 of the 129 cases mentioned above, and in a series of 400 cases analysed by Still 48 per cent. occurred in first-born children. But the condition has also been recorded as late as the fourteenth child of a family.

Symptoms. The usual story is that the baby has done well for the first few weeks, but then has begun to be sick after feeds—at first perhaps only once or twice a day, but soon being sick after every meal. This often gives rise to the idea that the mother's milk is not suiting her infant, and he is then promptly weaned on to some other food, with the result that for two or three days he seems better, but then the symptoms return and so another change is made in the diet. This sequence of events may be repeated several times, and with each change of food the symptoms temporarily improve, but they return with increasing severity, and the baby steadily loses weight.

The most striking symptom is *projectile vomiting,* and the appearance of this symptom is usually taken as marking the beginning of the disease. In some cases the vomiting at first takes the form of a gentle regurgitation, and only later assumes its projectile character, but when it is fully established the vomited material is often ejected with such force from the nose as well as the mouth as to spurt two or three feet from the infant. To begin with, the vomiting takes place after almost every meal, but as the disease progresses and the stomach becomes more and more dilated, there may be only one or two copious vomits each day. In such cases the vomitus may show a brownish discoloration, and there may be much mucus mixed with it. It is always strongly acid, and contains no bile.

Constipation is another prominent symptom, but not quite so constant as vomiting. Even when vomiting has been delayed until the third or fourth week the constipation may date from birth, and is thus often the earlier symptom. Occasionally constipation is not very pronounced, so that although the presence of forcible vomiting and constipation in an infant under three months old should always call to mind the possibility of pyloric stenosis, the presence of the former symptom alone should be enough to arouse suspicion.

Wasting is the third symptom of importance in the history of these infants. Generally a slow gain in weight takes place until the onset of vomiting, then for a short period the weight stands stationary before beginning to drop. When once the weight has begun to decline, the fall—in the absence of treatment—is fairly steady without intermission. As the disease progresses an almost typical facies gradually develops ; the face becomes thin and drawn, the expression becomes worried, and the brow becomes creased into transverse furrows.

Physical Signs. These are two in number, namely, visible peristalsis of the stomach and a palpable tumour at the pylorus. The latter is without doubt the more important, for while gastric peristalsis may be seen in other conditions at this age, such as pylorospasm and congenital duodenal stenosis, a palpable tumour at the pylorus is absolutely diagnostic of hypertrophic stenosis.

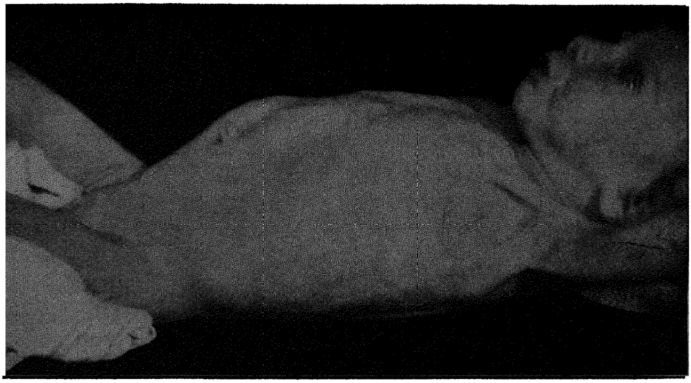

FIG. 12. Visible peristalsis of the stomach in hypertrophic pyloric stenosis. Infant aged 4 weeks.

In order that peristalsis may be seen, it is usually necessary to stimulate the stomach to contract by distending it with a feed. At the same time this keeps the infant quiet, so that observation can be made the more easily. The waves of peristalsis take shape at the left costal margin, and roll slowly across the epigastrium to fade away on the right side. In a well-defined case the waves give the appearance of a succession of golf-balls slowly passing across the upper abdomen, but in more advanced cases when the stomach is dilated the waves become broader and less obvious. Rarely peristalsis may also be made out passing in a retrograde direction from right to left.

When feeling for the pyloric tumour a proper technique must be adopted. The examination should be made while the infant is being fed, and the observer must be on the infant's left side, therefore the infant should be fed either from the mother's left breast or else by a nurse feeding him from his right side. The infant must be disturbed as little as possible, only the abdomen should be uncovered, the examiner's hand must be warm, and he should be on the same level as the infant, so that he can rest his hand comfortably across the baby's epigastrium. It is as well to lay the hand gently on the abdomen for a minute or so before beginning to palpate, otherwise the infant may squirm and make examination difficult. Then by pressing the fingers down over the region of the pylorus, half-way between the umbilicus and the costal margin at the outer border of the right rectus muscle, the tumour may be felt with the ball of the middle finger as a hard knotty lump about the size of a cob-nut.

An important feature of the tumour is that it undergoes periodic contraction and relaxation, and as it can only be felt during contraction it may be palpable one minute but have disappeared a minute or two later, and then after a brief interval it can be felt again. The tumour hardens during peristalsis, and that is one reason for having the infant fed during examination, but it may sometimes take ten or fifteen minutes before the pylorus becomes hard enough to feel.

Various circumstances may make the tumour unusually difficult to feel. It may be tucked up beneath a rather large liver ; or in a case of some weeks' duration when the stomach is much dilated the tumour may be pushed over towards the right flank, and also in this stage the stomach may be so distended with food as to envelop and hide the tumour. If such be the case, continued palpation will cause vomiting, which, by partly emptying the stomach, uncovers the tumour.

As an aid to diagnosis, the infant may be X-rayed after a bismuth meal. The films will show that the stomach has failed to empty even after three or four hours. X-ray diagnosis is, however, not to be recommended, for often these infants are so frail as to be much exhausted by the amount of disturbance entailed, while if the tumour has once been felt the diagnosis is assured, and radiography is unnecessary.

Pathology. The appearance of the stomach varies to some extent with the length of time that the symptoms have been present. The most striking feature at post-mortem examination is the remarkable hypertrophy of the musculature of the pylorus. The circular fibres are principally involved, and form a thick band of muscle which extends for about an inch along the pyloric antrum, terminating abruptly at the duodenum. The hypertrophy is best seen after the pylorus has been divided longitudinally, when the muscle mass covered by mucosa can be seen to project slightly into the lumen of the duodenum.

In the early stages the hypertrophied muscle terminates on the stomach side almost as abruptly as it does on the duodenal side, and the tumour is vascular. As time goes on the tumour becomes less vascular, and although the hypertrophy still terminates abruptly at the duodenum, it tends to spread back along the wall of the stomach and may nearly reach the cardia, indeed it has been noted as far back as the lowest inch of the œsophagus. It is at this stage that peristalsis is most marked and the tumour can generally be felt without difficulty. Eventually the tumour becomes avascular, white, of almost cartilaginous hardness, and shows little tendency to relax. The hypertrophy still terminates sharply at the duodenum, while the stomach, unable to overcome the obstruction at the pylorus, undergoes considerable dilatation. The effect of the hypertrophied muscle is to constrict the lumen of the pylorus, throwing the mucosa into longitudinal folds which may so block the passage that even water can scarcely be forced through it at autopsy.

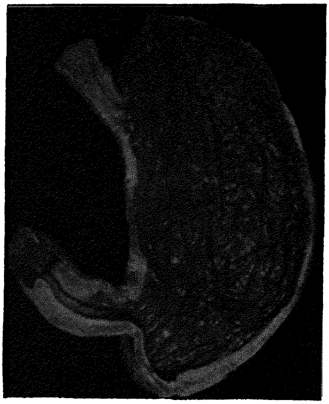

FIG. 13. Stomach from a case of hypertrophic pyloric stenosis showing the thick muscle tumour at the pylorus.

The cause of the condition is unknown. The excessive hardening of the tumour during contraction suggests a spasmodic element, as does the beneficial result that occasionally follows the use of anti-spasmodic drugs. The view propounded by John Thomson that the hypertrophy is the outcome of a perverted development of co-ordination of the nervous control of the pylorus seems to fit the facts as well as any. Presumably the peristaltic wave from the stomach reaches the pylorus at a time when the sphincter is contracting instead of relaxing. The striking linkage with the male sex points to the possibility of an inherited factor.

Diagnosis. The occurrence of forcible vomiting in an infant under three months of age, especially if it be accompanied by constipation, should always lead to an examination for gastric peristalsis and a pyloric tumour. The presence of a palpable pyloric tumour is of the greatest importance, not only because it occurs in no other condition, but also because when the disease is present the tumour, if properly examined for, can always be found. Constipation need not be present, peristalsis may not be visible, and Barrington-Ward has recorded an instance in which a tumour was palpated although there had been no vomiting, and the diagnosis of hypertrophic pyloric stenosis was confirmed at operation.

Two conditions likely to be confused with hypertrophic pyloric stenosis are congenital duodenal stenosis and pylorospasm. In the former a history of forcible vomiting and constipation dates from birth, while if the level of stenosis happens to be below the bile papilla the vomitus will contain bile. Gastric peristalsis can usually be seen, but palpation fails to reveal anything resembling the typical pyloric tumour.

Pylorospasm is more common in female infants. The symptoms begin at about the same time as in hypertrophic stenosis, and include forcible vomiting and often constipation. Gastric peristalsis may also be made out. In pylorospasm, however, there is no hypertrophy of the pylorus, and consequently no tumour to be felt, and herein lies the one important distinguishing feature which determines not only the diagnosis, but also the treatment. In pylorospasm there is no indication for operation, indeed, such cases usually do badly if they are treated surgically. They are best dealt with on the same lines as the medical treatment of hypertrophic pyloric stenosis.

Course and Prognosis. Death from starvation and progressive weakness at some time during the first three months of life is the usual end of untreated cases of pyloric stenosis. Occasionally the infant manages to strike a balance between the amount of food lost by vomiting and the amount which passes the pylorus, a balance which is just sufficient to keep the weight more or less constant. Under these circumstances the infant may struggle on until about fourteen weeks old, at which age the symptoms tend to clear up spontaneously, and at last the weight begins to rise and the child recovers. Once recovery has set in it proceeds as a rule smoothly without relapses. It is not understood why recovery should occur at about the fourteenth week; it is certainly not due to a disappearance of the muscle tumour, for this may remain palpable until the infant is six months old.

The outlook under treatment depends to some extent on whether surgical or medical measures are employed. In order to obtain the best results from surgery, operation must be undertaken as soon as the diagnosis has been made. It is a mistake to try medical treatment for a week or two and only to resort to surgery when medical means have failed to bring about improvement, for the infant is then weaker and less fit to undergo an operation. The surgical mortality rises as the interval increases between the onset of symptoms and operation, which emphasises the great importance attaching to early diagnosis. With either method the prognosis is better if breast milk is available.

The introduction of Eumydrin has undoubtedly increased the efficacy of medical treatment, but no hard and fast rules can be laid down to decide whether surgical or medical measures should be employed ; each case must be judged on its merits. The ideal case for medical treatment is the infant who is more or less holding his own at the time the diagnosis is made ; this would be indicated by a stationary instead of a falling weight, and by the passage of a small stool each day. For those who have been losing ground rapidly, or whose weight is below 7 lb., operation is to be preferred. It must be remembered that with surgical treatment the relief of symptoms is immediate, and in a successful case the infant quickly begins to regain weight and strength, while medical treatment may have to be persisted with for a month or more ; and in hospital practice the risk of intercurrent infections, such as bronchitis, otitis media, and diarrhœa, adds to the hazards of a long treatment.

The mortality with either method of treatment in hospital practice should be below 10 per cent. That it is undoubtedly much lower in private practice is due to several factors—the diagnosis is usually made earlier ; the nurse is able to give her whole attention to the one infant ; and the risk of ward infections is eliminated.

Surgical Treatment. Gastro-enterostomy, Loreta's operation of opening the stomach and passing dilators through the pylorus, and pyloroplasty have all been tried and abandoned in favour of Rammstedt's operation, in which the hypertrophied pyloric muscle is divided longitudinally down to the submucosa, so that the mucosa bulges up into the incision. The unstitched pylorus is then returned into the abdomen. Owing to the avascularity of the tumour, trouble from bleeding does not arise.

Although delay before operating is undesirable, enough time must elapse for proper pre-operative preparation to be carried out, which will often take about twenty-four hours. The stomach must be cleaned by being washed out, and if the first lavage is dirty, with brownish curds and a great deal of mucus, the wash-out must be repeated once or perhaps twice at intervals of twelve hours. A final lavage should be carried out an hour or two before the operation. It may be pointed out that the continual vomiting leads to a considerable loss of hydrochloric acid, which tends to throw the acid-base balance of the body towards the alkaline side, and this makes a severe and even fatal collapse more likely. On this account the stomach should not be washed out with sodium bicarbonate, which would tend to increase the state of alkalosis, but normal saline should be used instead. It is also the usual practice to give a subcutaneous infusion of normal saline with 5 per cent. glucose an hour or two before operation. If there has been a severe loss of weight, the injection of saline should be repeated after twelve hours, the operation being delayed meanwhile. Pre-operative preparation should be completed at least an hour before the time of operation.

The choice of anæsthetic is of considerable importance. For general anæsthesia gas and oxygen should be employed, the alternative being local anæsthesia, which would perhaps have preference were it not that it seems to weaken the resistance of the abdominal wound to local infections.

The post-operative management demands much care. Not only is the most skilled nursing required, but the nurse should be particularly experienced in the management of these delicate infants. As a rule feeds are started four hours after the operation, beginning with glucose water. Drachm doses are given hourly for four hours, then two drachms hourly for four hours. If breast milk is available, it should be given twelve hours after the operation, allowing half an ounce every one and a half hours. This should be increased to an ounce two-hourly after twenty-four hours, then two ounces three-hourly after thirty-six hours, and three ounces three-hourly after forty-eight hours, by which time the diet becomes normal for the age. It is a mistake to increase the diet more quickly than this, because a dietetic enteritis is readily set up in these infants. If breast milk is not available, its place may be taken by a half-cream dried milk or equal parts of milk and water suitably sweetened.

Various complications may follow the operation. Within a few hours the temperature may rise to 104° F. or higher, and a fatal hyperpyrexia may occur. It is quite common for the temperature to rise 2 or 3 degrees, and this has never been satisfactorily explained. It is possible that exudations escaping from the cut surface of the pyloric muscle into the peritoneal cavity may play a part. The temperature should be taken every hour for the first six hours or so, and if it rises dangerously the blankets should be raised off the infant, the warm bottles must be removed from the cot, and a tepid sponging may be necessary. If collapse occurs, ten drops of brandy every two or three hours may tide the infant over a critical period, and an injection of camphor (gr. ⅙) may be given in urgent cases.

Sometimes vomiting continues after the operation, and is often difficult to explain. Occasionally it is due to incomplete division of the pyloric muscle, and it is also likely to occur if the operation is undertaken before the stomach has been properly cleansed by the pre-operative gastric lavages. It should be dealt with by washing out the stomach once or twice with normal saline, after which the vomiting will generally cease. In other cases a troublesome diarrhœa may develop a few days after the operation, which necessitates a reduction in the diet and may lead to a further drop in weight, and unless the condition soon rights itself the infant may sink into a state of marasmus from which recovery is tedious, or which may prove fatal after three or four weeks. Small transfusions of whole blood will sometimes rally such cases when everything else has failed. When the operation is undertaken in an infant already severely wasted, the tissues may be so sluggish that the incision in the abdominal wall fails to unite, and when the stitches are removed the wound-edges fall apart. Two infants affected in this way eventually made a good recovery.

Medical Treatment. This is conducted along three main lines. The diet should be such as to give only a small curd in the stomach. Breast milk will achieve this most successfully, otherwise lactic acid milk, peptonised milk, or whey thickened by adding a teaspoonful of Benger's, Savory and Moore's, or Mellin's Food to every three ounces, may be given. Secondly, the stomach should be frequently washed out, as this will generally bring the vomiting quickly to an end. The wash-out should be done with normal saline, beginning with lavages twice a day until the vomiting ceases, afterwards repeating the lavage once a day for a week, and then gradually lengthening the interval between the wash-outs, provided that the vomiting does not return, until after perhaps three or four weeks they can be given up altogether. Thirdly, before each feed a small dose of some anti-spasmodic drug should be given. Liquor Atropinæ m. ¼ may be used, but the advent of Eumydrin (atropine methyl nitrate) has largely replaced other atropine salts, because its toxicity is so much less. A freshly prepared 1 : 10,000 solution is used, from 3 to 5 c.c. being given twenty minutes before each feed. Its administration should not begin until dehydration has been overcome by means of subcutaneous salines.

In addition to relaxing the pylorus, Eumydrin also relieves the colicky contractions of the stomach, which otherwise give rise to much discomfort and restlessness after feeds. The drug has also proved very useful in the treatment of pylorospasm (see p. 115). It may be necessary to continue it for a month or more.

CHAPTER VII

VITAMIN-DEFICIENCY DISEASES

BEFORE considering some of the diseases which result from deficiency of vitamins, a brief outline of our present knowledge of these substances is given. An appendix on pp. 717–721, shows the concentration of vitamins in some proprietary preparations.

VITAMIN A

Recent investigations have shown vitamin A to be an unsaturated alcohol related to carotene, which is a yellow crystalline pigment occurring in the body, and giving a yellow colour to certain natural fats ; it is also found in company with plant chlorophyll. Vitamin A cannot be synthesised in the body, but is present in milk fat, eggs, cod- and halibut-liver oil, and young green plants. There is experimental evidence to show that animals can convert carotene into vitamin A, and so carotene is regarded as pro-vitamin A.

The daily requirement of infants is about 2,500 international units (I.U.), and 4,000–6,000 I.U. for older children.

VITAMIN B

This is a complex vitamin, divided into vitamin B_1, the antineuritic vitamin, and vitamin B_2.

Vitamin B_1 (Aneurin : Thiamin). This has been isolated from rice polishings in the form of thiamine chloride, and can be prepared synthetically. The richest sources are the germ of cereals and yeast. The body is unable to synthesise it or store it in any quantity. The daily requirement in childhood is probably between 80 and 200 I.U.

Vitamin B_2. This is a complex substance containing five subdivisions, of which at least two concern man ; they are riboflavin and nicotinic acid.

Riboflavin. This is a pigment widely distributed in Nature, and may be obtained from such sources as milk (lactoflavin), egg (ovoflavin), meat, yeast, plants. In conjunction with phosphoric acid and a protein, riboflavin plays an important part in tissue respiration.

The daily requirement is thought to be from 1 to 3 mgm. The symptoms of riboflavin deficiency consist of fissures and crusting at the angles of the mouth and round the nostrils ; discolouration, fissuring and soreness of the tongue ; and vascularisation of the cornea.

Nicotinic acid. This may be obtained from nicotine or from such natural sources as yeast and liver, and can also be prepared synthetically. It is the anti-pellagra factor of the vitamin B complex.

VITAMIN C

(Ascorbic acid : Cevitamic acid)

This substance was first isolated in 1927 by Szent-Gyorgyi from the suprarenal cortex and from orange juice, and has since been prepared synthetically. The richest natural sources are the citrous fruits, black currants, strawberries and tomatoes.

For the prevention of scurvy the daily requirement of infants is not less than 20 mgm., and for older children 50–100 mgm.

VITAMIN D

(Calciferol)

The history of this vitamin is given in the introduction to Rickets. Owing to the formation of vitamin D in the skin under the action of the sun's rays, the amount that should be taken by mouth to prevent infantile rickets is difficult to determine. A daily allowance of 500–800 I.U. is usually sufficient.

VITAMIN E

This vitamin, having anti-sterility and anti-abortion properties, is contained in the embryos of seeds, and is prepared from wheat germ oil.

VITAMIN K

The discovery of this vitamin is due to the work of Dam. It is a derivative of naphthoquinone, and is present in green plants, particularly alfalfa (Lucerne) and spinach. Deficiency of the vitamin is associated with a low prothrombin content in the blood and a tendency to bleed, and it has proved useful in the treatment of hæmorrhagic conditions in the newborn. Several analogues, having an identical action, have been synthetised

VITAMIN P

(Citrin : Hesperidin)

This is a compound of the flavone type, closely associated with vitamin C, and considered to have a beneficial effect in diminishing the permeability of capillary walls (see p. 453.)

RICKETS

Introduction. Although rickets produces its most obvious effects upon the skeleton, it is actually a general metabolic disturbance and involves practically every system in the body. The disease is now known to result from a lack of irradiated ergosterol or vitamin D, which leads to a failure to absorb and retain the calcium and phosphorus of the diet. In order for the characteristic skeletal symptoms to develop it is also necessary that the child should be actively growing.

Until a few years after the Great War, there were two chief schools of thought as to the cause of rickets. On the one hand there were those who held the condition to be due to some dietetic error, for it was known that rickets was rare in breast-fed infants, and moreover could be cured by giving cod-liver oil; on the other hand there were those who maintained that environmental factors such as lack of light, bad ventilation and insufficient exercise accounted for the disease. It is not without interest briefly to trace the steps by which these two views have coalesced.

The discovery by Mellanby in 1919 that rickets could be produced experimentally in puppies if they were fed on certain diets marked the beginning of an intensive research into the dietetic aspect. Mellanby was led to conclude that rickets was produced by the absence from the diet of fat-soluble vitamin A. In the following year Hopkins showed that this vitamin could be partly destroyed by a process of heat and oxidation, and in 1922 MacCollum, by feeding rats on cod-liver oil which had been submitted to oxidation, was able to show that vitamin A really consisted of two substances, one capable of curing xerophthalmia (a condition known to be brought about by lack of vitamin A), the other capable of curing rickets.

Meanwhile the curative effect of direct sunlight was emphasised by Rollier in 1916, and three years later Huldschinsky showed that the same result could be brought about by exposing subjects with rickets to that portion of the ultra-violet spectrum having a wave-length of 300 $\mu\mu$.

Although rickets could be cured by ultra-violet radiation, and in cod-liver oil there was also a powerful anti-rachitic agent, it remained to correlate these facts. Zucker and others (1922) demonstrated that the anti-rachitic properties of cod-liver oil were contained in the non-fatty portion, consisting chiefly of a cholesterol-like substance, and in 1925 Steenbock and Black showed that this material, although not inherently protective against rickets, became so after exposure to ultra-violet radiation. Finally, in 1926, Rosenheim and Webster showed that the cholesterol-like substance was actually a closely allied sterol, namely ergosterol. Vitamin D, or Calciferol, the anti-rachitic vitamin, consists of irradiated ergosterol.

Ergosterol is normally present in the deeper layers of the skin, and becomes converted into vitamin D when the skin is exposed to sunlight or ultra-violet rays. Cod-liver oil, halibut-liver oil, egg yolk, and to a less degree milk, also contain irradiated ergosterol. It seems that milk, too, contains some inactive ergosterol, for its anti-rachitic value is increased after exposure to ultra-violet light. Ergosterol is also present in most animal and vegetable tissues in combination with cholesterol, and again, following a short exposure to ultra-violet radiations, is converted into vitamin D.

Etiology. *Distribution.* Rickets is widely distributed throughout the temperate zones, but is rare both in the tropics and in such a northerly country as Iceland. No race is immune from the disease, but there is no doubt that negro children who dwell in the temperate zones are particularly susceptible, probably because their natural pigmentation prevents a sufficient penetration of ultra-violet rays through their skin. Infants born prematurely are also prone to develop rickets, a fact which is generally attributed to the fœtus storing calcium salts chiefly during the last two months of intra-uterine life, so that a premature infant starts life handicapped by a deficient store of these salts.

Age. Rickets is unknown as a congenital condition, and can seldom be recognised clinically under four months of age, but may appear at any time from then until the end of the second year.

Diet. There is no doubt that the disease is very much less common in breast-fed infants than in those reared artificially, but breast milk is no guarantee against the appearance of rickets. Its anti-rachitic value depends upon the diet and habits of the mother, who should partake of such foods as milk, butter, and eggs for their vitamin D content, and should also get out of doors into the sunlight each day. Hess and Weinstock have shown that the anti-rachitic titre of breast milk can be raised to a higher level by direct irradiation of the mother than by supplementing her diet with cod-liver oil.

Even if breast feeding has been continued for the full nine months, a faulty diet after weaning, with excess of cereal food and little in the way of animal foods, may lead to rickets during the second year.

The process of drying or condensing milk does not entirely destroy its anti-rachitic value. Although it is the common practice at the present day, and a wise one, to add a small quota of vitamin D as a prophylactic measure to the diet of infants reared on these milks, it is probable that the increasing custom of letting infants lie out of doors for most of the day is at least as valuable a means of preventing rickets. The most severe instances of rickets occur when infants have been reared on a diet which contains much sugar and little fat, and for this reason feeding with the sweetened form of condensed milk is not to be commended—except for very short periods. It is even more inadvisable to feed an infant on a diet made up entirely from one of the many starch-containing foods which are intended as additions to a milk and water mixture.

Season. The incidence of rickets shows a seasonal variation, reaching a maximum in the late winter, after a period when, so far as this country is concerned, exposure to sunlight is a very chancy affair, and when incidentally the vitamin D content of such fresh foods as milk and eggs is also at a minimum.

Growth is another factor of etiological importance, for rickets will only develop if growth is taking place. This accounts for the fact that rickety infants are practically never wasted, they tend on the contrary to be fat, but flabby. Although marasmic infants so seldom appear rachitic, the other factors that underlie the disease may all be present, and then as soon as a recovery from the marasmus is made, and the infant begins to put on weight and grow, rickets is very likely to develop. The influence of growth is well illustrated by cœliac rickets, a condition to be described later. Children suffering from cœliac disease are profoundly wasted, and may remain so until they are four or five years old, becoming considerably stunted. It is not until they begin to gain weight and grow that cœliac rickets makes its appearance.

Pathology. If a longitudinal section is made through the growing end of a long bone from a case of rickets, the epiphyseal line is seen to consist of a broad pearly-grey translucent band of osteoid material—which is the matrix of bone in which calcium phosphate has not yet been deposited. The bone end is expanded laterally and is slightly cup-shaped, and the band of osteoid is correspondingly shaped, while its margins on both the diaphyseal and epiphyseal side are uneven and irregular. The adjacent diaphysis is also softer and more vascular than normal. The appearances differ very much from a normal bone-end, where the bone and epiphyseal cartilage approximate in a straight transverse line without the intervention of any visible osteoid.

The histological changes at the bone-ends can hardly be appreciated without first describing the appearances at the growing-end of a normal bone. As the normal epiphyseal cartilage is traced towards the epiphyseal line, the cartilage cells become arranged into columns lying in the long axis of the bone, and separated by columns of calcifying cartilage-matrix. On the other side of the epiphyseal line are young capillaries springing from the bone marrow and growing towards the cartilage. The capillaries are guided by the columns of calcified cartilage matrix against the cartilage cells, which are destroyed. The calcified columns then become broken up into masses around which the matrix of bone (osteoid) is formed. In health, osteoid is present only in small amounts, too small to be discerned macroscopically, and is soon converted into true bone by the deposition of calcium phosphate.

The striking feature about ossification in rickets is the entire lack of orderliness, owing to the lack of calcification. The cartilage cells are not forced into columns because the cartilaginous matrix fails to calcify, and so the capillaries from the young bone are not particularly directed against the cartilage cells, some of which may escape destruction and become enveloped in the osteoid. Nevertheless, so long as growth continues, most of the cartilage cells are being steadily replaced by a bony matrix which also fails to calcify, remaining as osteoid. It is this accumulating osteoid which gives the characteristic broad, wavy and soft epiphyseal line to the rachitic bone.

As the rachitic process heals, calcium is first of all deposited in the cartilage matrix, which soon reduces the cartilage cells into order. The osteoid layer becomes calcified, any cartilage engulfed in the osteoid tissue slowly disappearing.

The growth of bone from the periosteum is also interfered with, soft osteoid tissue being produced along the shaft of the bone. As soon as healing begins, this becomes converted into true bone by the deposition of calcium phosphate so that eventually the rachitic bone may become a thicker and stronger bone than would normally have been the case.

Chemical Pathology. It has been pointed out that the essential disturbance of the skeleton in rickets lies in the failure of calcium phosphate to be deposited at the growing ends of the bones.

Theoretically there are several ways by which such a failure might come about. The diet might contain an insufficient amount of calcium and phosphorus, or the absorption of these materials from the bowel might be interfered with. If absorption were satisfactory, there might be yet a failure in the mechanism at the bone ends by which calcium and phosphorus are withdrawn from the blood and deposited in the osteoid tissue.

It is known that the deposition of calcium phosphate will only take place in the presence of a ferment—phosphatase, but estimations of phosphatase in infants with rickets show it to be present in amounts above the normal, so that it would seem that the mechanism for the withdrawal of calcium phosphate from the blood to the growing bone is not impaired. As to any deficiency of calcium and phosphorus in the diet, experience shows that the additions of these materials to an otherwise rachitogenic diet will not prevent or cure rickets.

Estimations of the blood calcium and blood phosphorus in active rickets show either or both of them to be reduced. In the majority of cases the blood calcium is within normal limits (9 to 11 mgm. per 100 c.c.) but the blood phosphorus (normal childhood value 4 to 6 mgm. per 100 c.c.) is reduced, sometimes to as low a figure as 2 mgm. per 100 c.c. This is spoken of as the " low phosphorus type " of rickets. Less often the blood phosphorus is normal while the blood calcium is reduced, in some cases being as low as 6 mgm. per 100 c.c., and in one case under the author's care the calcium was down to 4 mgm. per 100 c.c. It is this type of rickets, known as the " low calcium type " which is liable to be accompanied clinically by other manifestations of a diminished blood calcium such as tetany, laryngismus stridulus, and convulsions—a triad of symptoms grouped together under the name "spasmophilia ". Occasionally both phosphorus and calcium values in the blood are reduced below normal.

A feature of the metabolism in rickets is the excessive loss of calcium by way of the stools, the loss in the urine being inappreciable. This points to a defect in the absorption of calcium from the intestine, although it has been suggested that the calcium is absorbed and is then re-excreted in abnormally large amounts into the intestine. The excretion of phosphorus in the urine is also excessive, in fact the loss of phosphorus is relatively greater than the loss of calcium. There is no doubt that the proper absorption and interaction of calcium and phosphorus can only be effected if they are available in a balanced proportion, excess or paucity of the one affecting the utilisation of the other.

Howland has expressed this in its relation to rickets by saying that when the product obtained by multiplying together the blood calcium and blood phosphorus values (measured as mgms. per 100 c.c.) falls below 30, active rickets is present, while rickets is probably present if the figure is below 40. The normal figure should lie between 40 and 60.

The exact manner in which vitamin D brings about an improvement in the calcium-phosphorus metabolism is not absolutely certain, but it is most likely that it indirectly facilitates the absorption of these materials from the intestine, and their retention in the serum, by increasing the capacity of the blood to carry them.

Symptoms. Increasing knowledge concerning the composition of vitamin D and the natural sources from which it can be obtained has made rickets one of the most easily preventable of diseases, with the result that it is rapidly becoming an uncommon condition, and in fact the gross skeletal deformities of advanced rickets are already a rarity.

The onset is gradual, and definite signs of the disease can seldom be made out before the fourth month. As a rule the infant is plump, and may at first sight look healthy enough, but there is likely to be a history of increasing restlessness and irritability, and of excessive sweating, particularly about the head. The most pronounced changes are those that affect the skeleton, and we may therefore consider these first.

The Skull. Ossification of the rachitic skull proceeds slowly and irregularly. The anterior fontanelle remains larger than the age of the infant would warrant, and instead of closing at about the eighteenth month, may remain open into the second or even third year. The irregularity of ossification is also seen in the heaping up of young bone into bosses round the centres of ossification of the frontal and parietal bones. Under the name of "Parrot's nodes," these bosses were originally regarded as evidence of congenital syphilis, but they are now more rightly regarded as rachitic. The sagittal and coronal sutures, radiating from the four corners of the anterior fontanelle, and lying as furrows between the bosses, give to the skull the appearance of a hot-cross bun.

In other parts of the skull small areas of thinning and rarefaction occur, particularly over the lower parts of the parietal bones and along the occipito-parietal sutures. Careful palpation is needed to discover them. The observer should hold the infant's head between his palms, and then gently press upon the skull with his finger tips. The bone in the thin areas can be indented just as one can indent a tennis-ball, springing back when the pressure is released. To this sign is given the name " craniotabes " and it affords valuable evidence of rickets, being one of the earliest manifestations. It also occurs in the thinned-out skull of hydrocephalus, and is present in the rare condition of osteogenesis imperfecta. When rickets has been present for several months, the skull gradually assumes a typical " square " shape, becoming flattened on top, and with a high bossed forehead. The head then appears rather larger than normal, although it is not likely to be confused with the globular head of hydrocephalus.

The eruption of the teeth is often delayed, and the first teeth may not appear until well into the second year, but seeing that in perfectly healthy infants the date of appearance and order of eruption of the teeth is subject to considerable variation, delayed eruption should only be looked upon as confirmatory evidence of rickets when there are already other criteria for making the diagnosis. In some rickety children the teeth of the first dentition appear strong and healthy, and this is reasonable enough when one remembers that the first dentition is to a large extent calcified before birth ; but more often the teeth are poorly formed and tend to premature decay, which accords with Mellanby's experimental evidence that a high cereal diet poor in vitamin D (such as is often the diet of rickety children) tends to promote dental caries. Rickets is also likely to exert a harmful influence on the teeth of the second dentition, for at the age when rickets is actiye, these teeth should be undergoing calcification. Another effect of rickets is to cause the teeth to be too crowded. This is due in part to the impaired growth of the jaw, and in part to the chronic naso-pharyngeal catarrh which is so common in rickets, and which, by obstructing nasal breathing, assists in the formation of a high and narrow palate.

The Thorax. Beading of the ribs is one of the most common and earliest signs of rickets. The beads occur at the junction of the ribs with the costal cartilages, and are most developed on the fourth, fifth, and sixth ribs just external to the nipples. In severe cases they are sufficiently marked as to be easily seen, and constitute the " rickety rosary ", but such large beads are not common at the present day. In slighter cases they can only be made out by palpation ; normally the junction of rib and costal cartilage can scarcely be detected, but in the rickety child the junction is easily felt as a small knob. The beads are larger on the internal surface of the thoracic wall, and at autopsy the surface of the lungs may be indented by them.

The ribs are also softened, and tend to yield before the negative pressure within the chest, and so the thoracic wall may become flattened at the sides while the sternum becomes pinched forwards into a " pigeon-breast ", and in severe cases a vertical furrow may form down each side of the sternum. The ribs over the liver on the right side and over the stomach on the left tend to splay forwards, forming with the chest above a horizontal groove—Harrison's sulcus—at the level of the upper border of the liver.

Mention may be made here of the soft and springy chest wall of some children who show no trace of rickets. Generally these children are rather puny, and their unusually resilient chest wall is in keeping with their poor physique. Clinical and radiological investigation shows, however, no evidence of rickets, nor does anti-rachitic treatment add any firmness to their chest wall, which is likely to remain soft for several years.

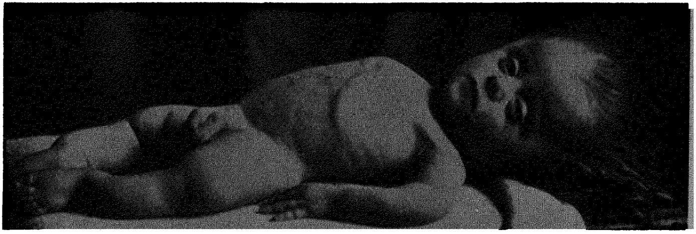

FIG. 14. Severe rickets with bossing of the skull, rickety rosary, enlarged epiphyses, and curvature of the long bones.

Spine and Pelvis. Changes in the spine consist of kyphosis, and sometimes scoliosis as well, due partly to softening of the vertebræ, but principally to laxity of the muscles and ligaments which should brace the spine. The kyphosis is an exaggeration of the normal dorsal curve. The pelvic deformities consist of a projection forwards of the sacrum, with a pinching-in of the lateral walls. Although the pelvic changes occur much less often at the present day owing to the early stage at which most cases come under treatment, they form one of the most serious consequences of rickets in a female child owing to the difficulties they may cause in later life during parturition.

Long Bones. Enlargement of the epiphyses of the long bones is a common feature of moderately advanced rickets, and is best seen at the ankle and wrist, but when the infant is plump and the rachitic process is still in an early phase it is often difficult to decide whether swelling of the epiphyses is actually present. The matter can, however, be settled at once by an X-ray examination, in fact radiological changes can often be made out at the lower end of the ulna at a very early stage of the disease, before the rest of the skeleton shows any alteration. On this account an X-ray of the wrist is generally looked upon as a valuable criterion when making an early diagnosis (see Plate I, Fig. A). The X-ray changes consist of a splaying out and hollowing of the ends of the long bones, while the line of ossification is irregular and fluffy instead of being an even transverse line, and the bone behind the epiphyseal line is rarefied. In cases of some standing one or more transverse lines of more dense bone can often be seen lying parallel and close to the epiphyseal line, as though the rachitic process had made previous attempts at healing.

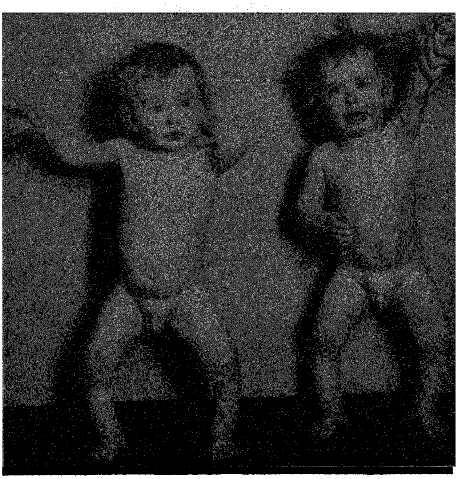

FIG. 15. Rachitic bow-legs in twins aged eighteen months.

In addition to changes at the epiphyses, bending of the long bones is likely to occur, due in part to the pull of the muscles on the softened bones and in part to the gravitational pull of the weight of the limbs. The direction of bending is as a rule an exaggeration of the normal curve, the femur bows forwards and outwards while the lower ends of the tibia and fibula curve backwards and inwards, giving an appearance of bandy legs.

The weight of the child while crawling may also give rise to bowing of the bones of the arm. On account of their softening, the long bones are more liable to fracture, the usual form being a greenstick fracture or crack without rupture of the periosteum.

Of symptoms outside the skeletal system, *anæmia* is one of the most common. It is hypochromic in type, the hæmoglobin being relatively more reduced than the number of red cells, and so the colour index is low. A mild degree of leucocytosis is often present, because rickety infants are more liable to infections than are healthy children.

Loss of muscular tone and increased laxity of the ligaments is another common feature, which often makes it possible for the limbs to be put into contortionist attitudes. Delay in sitting up and in walking is entirely due to this loss of tone, and is not to be attributed to any mental deficiency, which is not a symptom of rickets. The abdominal distension, or pot-belly, which characterises many rachitic infants results partly from the loss of tone in the muscles of the abdominal wall and partly from the gaseous distension of the intestine from excessive fermentation of carbohydrate, especially cereals, which so often forms a large part of the child's diet. The recti muscles become thin and separated, and this is well seen when the child attempts to sit up, the abdominal contents bulging forwards between the recti like a ventral hernia. The spleen can often be felt about a finger's-breadth below the costal margin.

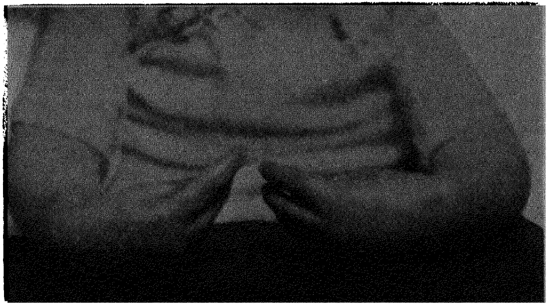

FIG. 16. Showing the characteristic position of the hands in tetany.

Spasmophilia

Under this name are grouped the nervous disorders—tetany, laryngismus stridulus, and convulsions—which arise when the calcium content of the blood is reduced, and therefore may be expected in the low-calcium type of rickets. The occasional appearance of tetany in the newborn has been considered on p. 24.

Tetany. The characteristic evidence of tetany consists of spasm of the muscles of the hands and feet—carpo-pedal spasms—although other groups of muscles may sometimes be affected. During the spasm, the thumb is inturned across the palm, the hand is flexed at the wrist, and the metacarpophalangeal joints are flexed while the inter-phalangeal joints are extended—the so-called "accoucheur's hand". The feet are turned downwards and inwards and the toes are flexed. The spasms, which are at first painful, may last for a few minutes or for several hours, and any attempt to alter the position of the extremities is likely to cause considerable pain. Carpo-pedal spasms are evidence of active tetany, but before they appear there are three signs by which latent tetany can be demonstrated. They are Chvostek's sign, Trousseau's sign, and Erb's sign.

Chvostek's sign. This is detected by tapping lightly over the branches of the facial nerve as they spread across the ramus of the mandible. In the presence of latent tetany, the facial muscles supplied by the particular branch of the facial nerve which has been stimulated give a momentary twitch, and in this way the eyelid, the alæ nasi, the corner of the mouth, and the platysma myoides may in turn be made to contract. Chvostek's sign is of considerable value in early rickets, as it may be present before rachitic changes in the skeleton are at all obvious.

Trousseau's sign. This consists of forcing a carpal or pedal spasm by temporarily compressing the limb and obstructing the venous return. It is less constant than Chvostek's sign.

Erb's sign consists of an increased excitability of the muscles to the galvanic current, and is of value inasmuch as it affords the earliest evidence of tetany.

Laryngismus Stridulus, or child-crowing, is due to a spasmodic closure of the glottis. In a severe attack the infant suddenly stops breathing and may turn blue in the face; after thirty seconds or so the spasm partly relaxes, and the rush of air into the chest through the still narrowed glottis gives rise to an inspiratory whoop or crowing sound. Often there is no preliminary pause in the breathing nor cyanosis, and the infant is simply heard to make a high-pitched cooing sound during inspiration, particularly if he is at all excited or irritable, laughing or crying. Many such attacks may occur in the course of a day, but they are seldom dangerous; death from suffocation in a severe attack has, however, been recorded.

Convulsions. The convulsions of spasmophilia do not differ in appearance from epileptic seizures. The occurrence of fits in a child between six months and two years of age should always prompt a careful examination both for rickets and for other evidence of spasmophilia, for spasmophilic fits are only temporarily checked by sedative drugs, and will certainly return until active treatment of the rickets has been undertaken, and the blood calcium has risen.

Complications. The two most common complications of rickets are infections of the respiratory tract and diarrhœa. Chronic infection of the upper air passages, especially the adenoids, frequently accompanies rickets, and may lead to much nasal catarrh or to otitis media. Even more important is the obstruction to the entry of air into the chest caused by the enlarged adenoids, for this aids considerably in producing the deformities of the chest wall. Attacks of bronchitis are frequent, and broncho-pneumonia often supervenes. The low resistance of the rachitic child against infections in general, coupled with the defective movements of the chest wall, makes broncho-pneumonia a dangerous complication.

Diarrhœa, due as a rule to the excessive amount of starchy foods in the diet, is not only a common complication, but is one which is likely to continue long because of the chronic distension and atony of the intestine. These infants withstand diarrhœa badly, and what in a healthy child might be expected to be a fleeting attack may in a rickety child prove rapidly fatal.

Diagnosis. When the various skeletal deformities are well developed the diagnosis should not offer any difficulty, but at the present day rickets is a rapidly disappearing disease, and severe examples of the condition are seldom seen. The diagnosis has generally to be made while the disease is in an early stage, and it is then more than ever important to bear in mind that rickets is a general disease, for if one suggestive sign appears, careful examination will generally furnish corroborative evidence in support of the diagnosis. A diagnosis of rickets based upon a solitary physical sign will be more often wrong than right.

Of early symptoms, restlessness, head sweating, and irritability are so common in other conditions that, by themselves, they are not sufficient to establish the diagnosis. Irritability must not be confused with actual tenderness of the bones ; rickets is not a painful disease, and if an infant's limbs are painful when handled he is more probably suffering from infantile scurvy, or, if below six months of age, from syphilitic epiphysitis. The two earliest skeletal changes are craniotabes, which may be present as early as four months, and beading of the ribs, both appearing often

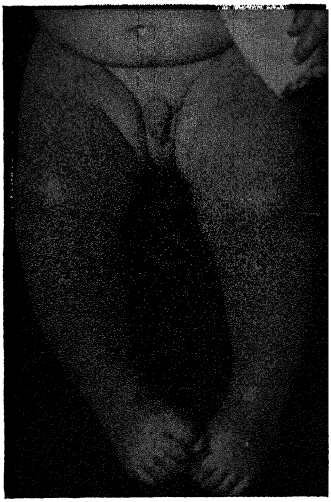

FIG. 17. "Physiological" curve of the legs in a baby one year old. There were no clinical or radiological signs of rickets.

before radiological evidence of the disease can be obtained. By the time the child is nine months old the anterior fontanelle may be noticeably big, and also by this time the delay in dentition will add support to the diagnosis. Bossing of the skull and enlargement of the epiphyses at the wrists and ankles appear at about this time, while bending of the bones generally develops during the second year.

The mere fact that an infant seems to have "bandy legs" does not necessarily justify a diagnosis of rickets. Curvature of the tibiæ is a relatively late sign of rickets, and when it is present there are always other manifestations of the disease. It is a common error to mistake for rickets the slight outward bowing of the leg which is apparent in many perfectly healthy infants, and which is due in part to the accumulation of fat on the outer side of the leg and in part to the natural "varus" position of the feet. This "physiological" curvature of the legs disappears during the second year when the child learns to walk, and it is a mistake to try and keep such an infant off his legs. The physiological curve is readily distinguished from the pathological curve of rickets, for the former is a gentle curve affecting nearly the whole length of the leg from the knee to the ankle, while the rachitic tibia is bowed chiefly in the lower third, and the lower end of the tibia is bent backwards as well as inwards. This common source of error will be avoided if other signs of rickets are insisted upon in addition to bandy legs.

The effects of rickets may be more prominent in other systems than the skeletal. Anæmia may be altogether out of proportion to other symptoms, and it may require six or eight weeks of treatment before the sallow complexion improves. In others the laxity of the muscles and ligaments may be profound, calling to mind the rare condition of amyotonia congenita.

X-ray changes at the ends of the long bones may be a useful aid to diagnosis in early and doubtful cases, and such changes are best seen at the wrist. But changes visible on the X-ray film are not always the earliest evidence, for they may be preceded by craniotabes and by alterations in the serum calcium and phosphorus.

Prognosis. Uncomplicated rickets is seldom a fatal condition ; its danger lies in weakening the child's resistance against infections, the immediate cause of death being usually bronchopneumonia or diarrhœa. The prognosis of the infectious fevers, particularly measles, whooping-cough, and influenza, is made much worse when they are complicated by rickets.

It is only in the more severe cases that bony deformities survive for long after the rachitic process has healed. Beading of the ribs and enlargement of the epiphyses generally disappear completely, and so as a rule does the bending of the long bones, but if treatment is delayed until the child is three or four years old, these deformities are likely to persist, and the child grows up stunted, with perhaps spinal curvature, a deformed thorax and pelvis, and either bow-legs or knock-knees. Certain parts of the skeletal are more likely to remain misshapen than others. Harrison's sulcus and eversion of the lower ribs is not infrequently seen in the later years of childhood, and the remains of bossing of the skull may be detected for many years.

Treatment. The principle underlying the treatment of rickets is to ensure a sufficient supply of vitamin D, either by giving cod-liver oil, halibut-liver oil, or pure irradiated ergosterol, or else by exposing the child to direct sunlight or to ultra-violet light. While it is possible to cure rickets by either of these methods alone, in practice it is desirable that the cure shall be as rapid as possible, and therefore both methods should be combined.

With regard to vitamin D given by mouth, the question will arise whether this should be given in the concentrated form of the pure vitamin or in its natural state as it occurs in cod- and halibut-liver oil. Our knowledge of vitamins goes to show that only small amounts of any particular vitamin are required in order for it to exert its optimum effect. Overdosage with vitamin D in animals is known to have deleterious effects, chiefly by causing calcification of the kidneys and arteries, and in children it is certainly possible to maintain the calcium and phosphorus in the blood at a level well above the normal figure by giving excessive doses of the vitamin, with a consequent danger of renal and arterial calcification. It must also be remembered that cod-liver oil, quite apart from its vitamin content, has a definite food value as a source of fat, and for these reasons the natural source of the vitamin is to be preferred to the pure concentrate. On the other hand rickety infants are occasionally unable to tolerate adequate amounts of cod-liver oil, developing diarrhœa and sickness or entirely refusing it because of its taste, and then resort must be made to one of the concentrated preparations.

Cod-liver oil is most conveniently given as an emulsion containing 50 per cent. of liver oil ; a teaspoonful of the emulsion should be prescribed four times a day. The other ingredients of the emulsion will consist of mucilage, a preservative, and fractional doses of various essential oils to act as flavouring agents. Of the many proprietary brands of cod-liver oil emulsion, any that contain 50 per cent. of oil may be regarded as reliable. It may be pointed out that the numerous preparations of cod-liver oil with malt contain a much lower percentage of the fish oil, and so are less strongly anti-rachitic, nor is the malt always desirable—particularly in the youngest instances of rickets. Cod-liver oil and malt finds a greater value in slightly older children who are in need of extra nourishment.

If it is decided to give a more concentrated form of vitamin D such as halibut-liver oil or even pure irradiated ergosterol, of which there are now several proprietary preparations, a dose of five drops thrice daily should be given, which will be more than the equivalent of drachm doses of cod-liver oil.

Treatment by direct exposure to the sun's rays is only of practical value in this country during the summer months. During the winter months, artificial light treatment should be carried out by exposing the infant to the rays from a carbon-arc or mercury vapour lamp. Care must be taken to eliminate chilling draughts. Generally the lamp is placed at a distance of about three feet from the child, exposures being made daily or every other day, the length of time of exposure being gradually increased from a minute up to a quarter of an hour as the skin slowly pigments.

The progress of healing should be recorded by X-raying the wrists at fortnightly intervals ; after the first fortnight improvement should be obvious, the epiphyseal line becoming denser and more regular in outline.

Other preparations have been used in the treatment of rickets,

PLATE I.

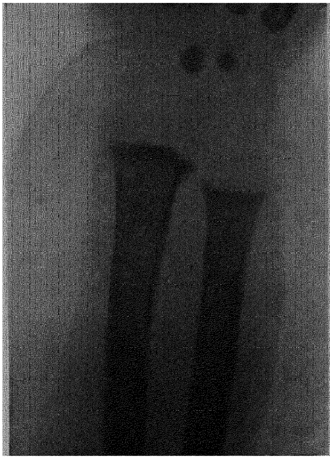

Fig. A. Early rickets. The lower end of the ulna is broadened, hollowed and irregular.

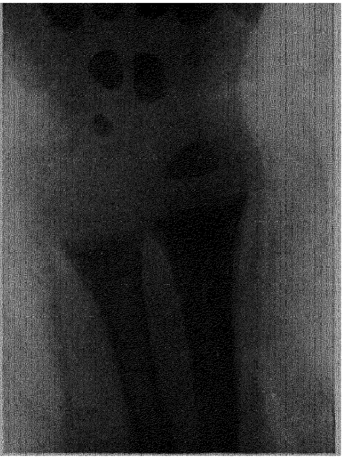

Fig. B. Severe rickets in a child aged two years. The lower end of the radius and ulna is broadened and concave, and the epiphyseal line has a spiculated outline.

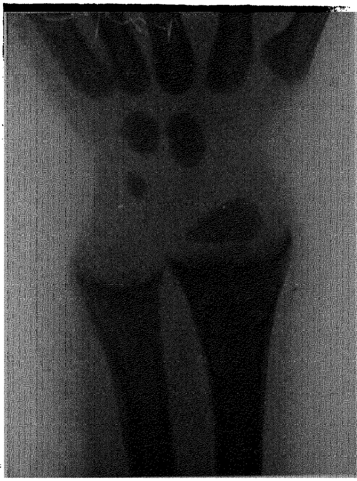

Fig. C. Rickets, showing an early stage of healing. The lower end of the radius and ulna shows a dense line due to the deposition of calcium salts. Same child as Fig. B.

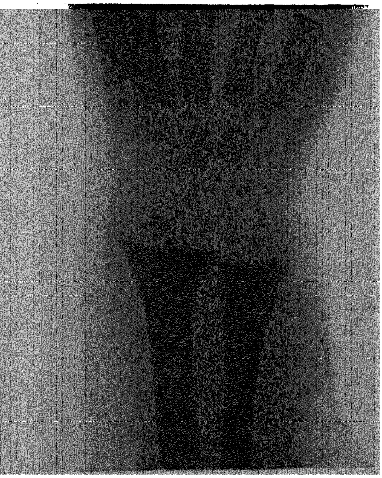

Fig. D. Lower end of radius and ulna showing almost complete healing of rickets. Infant aged eight months.

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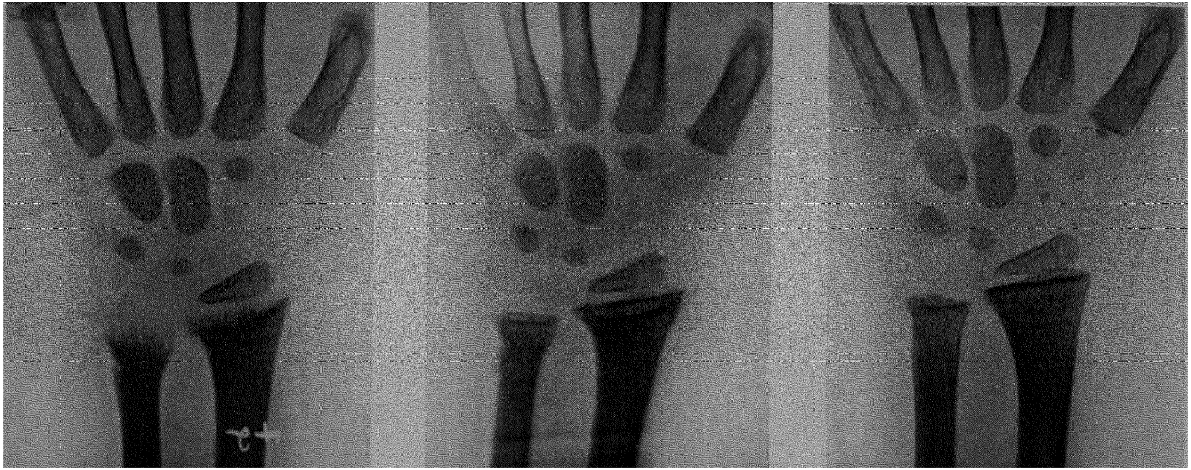
PLATE II.



Renal rickets in a boy aged four years, showing the bones undergoing healing. Treatment consisted of giving sodium bicarbonate 50 gr., sodium citrate 50 gr., calcium gluconate 90 gr., and vitamin D 20,000 I.U., daily over a period of six months. The boy died later of chronic uræmia.

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especially phosphorus and calcium salts, but provided that the diet contains an adequate amount of milk, and after nine months of age such additional sources of phosphorus and calcium as butter, egg-yolk, and green vegetables, there will be enough of these salts to cure rickets in the presence of vitamin D. Without this last factor, merely raising the intake of phosphorus or calcium makes little difference. Iron (ferri et ammon. cit. gr. 3 to 5 ter die) should be given when anæmia is a prominent symptom.

When rickets is complicated by tetany the serum calcium must be raised as quickly as possible. In addition to the usual anti-rachitic treatment 10 c.c. of a 10 per cent. solution of calcium gluconate should be given daily by intramuscular injection. In more urgent cases an initial dose may be given intravenously.

The question of keeping the child " off his legs " often crops up. As a rule a child with active rickets is so flabby and atonic that he does not make much attempt to walk, but in the more severe cases or when the child has already begun to crawl or walk, it is as well to prevent him from moving about during the first fortnight of active treatment, but after then the bones will be a good deal stronger, and the child's activities need not be curtailed. The best way to keep the child from using his legs is to let him wear long lateral splints from the knees, projecting two or three inches below the feet. When bowing of the legs has already occurred, splints may be applied down the outer side of the leg to prevent further deformity, but they should be ordered with this object in view and not in the hope that by firm bandaging the softened bones can be forced straight. It is a well-recognised fact that the curved bones of rickets tend to straighten during the year or so that follows healing, and, because of this, orthopædic operations to correct the alignment of the bones should be delayed in order to give Nature the opportunity of remedying the deformity herself. Massage during the healing stages is often valuable, helping the child quickly to regain the lost tone of the muscles and ligaments.

Prevention. Rickets is so much less common in breast-fed infants than in those fed artificially that the insistence upon breast-feeding for the first nine months may be justly regarded as a sound prophylactic measure. The disease most commonly occurs in children who have been reared on the sweetened form of condensed milk or have been given a diet containing cereal and unconverted starch from an early age, and in this connection the statement made in an earlier chapter that foods containing starch should not be given to infants under six months of age may be repeated here. But it is not only at this age that starchy foods may be harmful. Towards the end of the first year when the diet is expanding from milk to more solid food, too much cereal food such as bread, potato and biscuit is apt to be given to the exclusion of milk, butter, eggs, and green vegetables, particularly among the poorer classes, and this is likely to lead to rickets.

Except in the warm summer months, it is a good rule to add vitamin D to the diet of infants and children up to at least the end of the second year. A teaspoonful of cod-liver oil twice a day will be sufficient, or alternatively ten drops (5 minims) of halibut-liver oil once a day will serve instead.

A great deal may also be accomplished by keeping the infant out of doors as much as possible. When the weather permits he should be taken out in the morning and afternoon, and in the summer months should spend most of the day out of doors. It has been shown that even the reflected sunlight from clouds has a beneficial effect in preventing rickets.

The increased likelihood of rickets developing in infants born prematurely, and also in those who are recovering from any wasting condition, must be particularly borne in mind so that preventive measures may be undertaken as soon as possible.

Late Rickets

This term is applied to instances of rickets that begin in the middle or late years of childhood. The ordinary infantile type of rickets may occasionally continue into the third or fourth year if the diet has been grossly neglected and the child has for some reason been unable to get out of doors, but such cases are exceptional, nor should they be called " late rickets ", seeing that the rachitic process will certainly have begun at some time during the first two years. Most children by the time they are two years of age receive a sufficiently varied diet including milk, eggs, butter, and animal fats, to ensure an adequate supply of calcium, phosphorus, and vitamin D, and also spend much of their time out of doors, which prevents them from developing rickets. At the end of the 1914–18 War, however, the factory population in the Central European countries suffered from a diet which was deficient in the foods just mentioned, and many instances of rickets occurred in children up to the age of puberty, while at the same time examples of osteomalacia, or bone-softening, were met with in young adults. These diseases were undoubtedly due to lack of those foods which normally supply the salts necessary for the calcification of bone, coupled with an indoor life in the factories.

It is now generally accepted that osteomalacia in the adult is the counterpart of rickets in the child, and it is of interest to note that the incidence of the former condition in India lies chiefly among the girls and women who, living under the Purdah system, dwell indoors or veil themselves when they go out, and so avoid exposure to sunlight.

Late rickets is a rare disease in this country. It occurs as a rare complication of cœliac disease, and in association with chronic interstitial nephritis.

Cœliac Rickets. It has already been pointed out that growth must be actively taking place in order for rickets to develop, and as one of the clinical features of cœliac disease is severe stunting and a stationary weight, the complication of rickets cannot arise until such time as the cœliac condition has recovered sufficiently for growth to begin again. According to Parsons, cœliac rickets does not occur unless the cœliac disease has been severe, and recovery has been delayed until the child has reached seven years of age. Before this age, the bones simply become rarefied. The cause of cœliac rickets is not far to seek, for the essential feature of cœliac disease is an inability to absorb fat from the intestine, with the result that the child is starved of phosphorus and calcium salts and the fat-soluble vitamins.

The bone changes are the same as in ordinary infantile rickets if allowance is made for the difference in age. The fontanelle will be closed, but beading of the ribs and enlargement of the epiphyses appear, genu valgum is common, and in severe cases multiple fractures may occur.

Treatment is made difficult because the children cannot tolerate fat in the diet, and so it is difficult to supply an adequate amount of calcium and phosphorus. As soon as recovery begins to set in, an attempt should be made to prevent the development of rickets by exposures to ultra-violet light, and by giving five drops of irradiated ergosterol twice a day. Even if bony changes have appeared, persistent treatment on these lines will generally bring about a recovery.

Renal Rickets. The first clinical description of renal rickets was given by Morley Fletcher in 1911, although the association between rachitic deformities and albuminuria had been previously noted. Although all the skeletal deformities of infantile

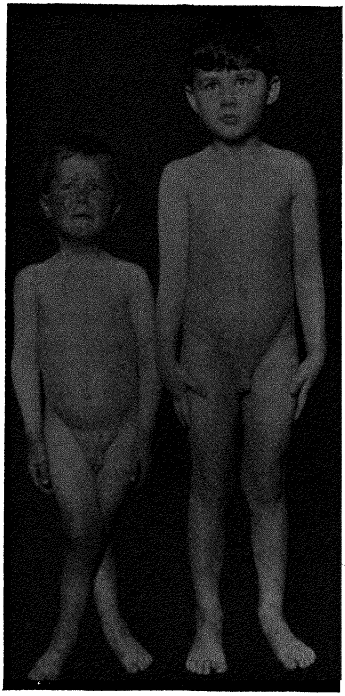

FIG. 18. Renal dwarf (on left) aged six years, standing with a normal boy of the same age. Note the genu valgum.

rickets may be reproduced in renal rickets, including a rickety rosary, enlargement of the epiphyses, and bending of the long bones, two outstanding features are a progressively increasing degree of knock-knee and a considerable stunting of growth, the last to such an extent that the children are spoken of as " renal dwarfs ". In addition to the skeletal deformities, the customary signs of fibrosis of the kidneys, such as great thirst, polyuria, a sallow and dry skin, and a variable amount of albumin and a few casts in a pale urine of low specific gravity, are also present.

The bone changes may begin at any time during the first two decades, most commonly between eight and twelve years of age, although dwarfing is often present for some years before the rachitic changes appear. X-ray examination of the bones shows a condition at the epiphyseal line which is indistinguishable from ordinary infantile rickets (see Plate II), and histological examination of the bone ends shows a state of affairs identical with infantile rickets. The shafts of the bones show either rarefaction or have a stippled or mottled appearance, which Langmead and Orr [1] have attributed to osteitis fibrosa, caused by para-thyroid hypertrophy arising as a result of the nephritis and low blood calcium.

The cause of renal rickets is not properly understood. It seems quite clear that these children do not suffer from any lack of vitamin D. Chemical investigations show that in the majority of cases the level of inorganic phosphorus in the serum is raised well above the normal figure (4 to 6 mgm. per 100 c.c. serum), rising sometimes as high as 10 to 14 mgm. per 100 c.c. The reason for this is the inability of the diseased kidneys to excrete

[1] Langmead, F. S., and Orr, J. W., *Arch. Dis. Child.*, 1933, **8**, 265.

phosphates in normal amounts. The serum calcium may be unaffected, but is usually lowered, sometimes to a remarkable extent; in the child whose X-rays appear on Plate II, the calcium was down to 4·8 mgm. per 100 c.c. serum, the normal figure being 9 to 11 mgm. Parsons has expressed the view that the failure of calcium phosphate to be deposited in the bones is attributable to the disturbance of the calcium-phosphorus ratio in the serum. Even when the calcium is as low as in the case mentioned, tetany is unusual, and this is probably due to the degree of acidosis that accompanies chronic nephritis.

The ultimate prognosis is hopeless. Several attacks of uræmia may be survived, but eventually one of them proves fatal, usually at some time between ten and twenty years of age. Ordinary anti-rachitic measures have no appreciable effect on the skeletal changes, although light orthopædic apparatus may be worn to check the increasing knock-knee. Recently Graham and Oakley [1] have reported considerable bony improvement by giving a combination of large doses of alkalies with big doses of vitamin A and vitamin D. The bones in Plate II healed under the influence of alkalies (100 gr. daily), calcium gluconate (90 gr. daily), and a big dosage of vitamin D (20,000 I.U. daily). Unfortunately any improvement in the skeletal condition brings no amelioration to the prognosis of the nephritis.

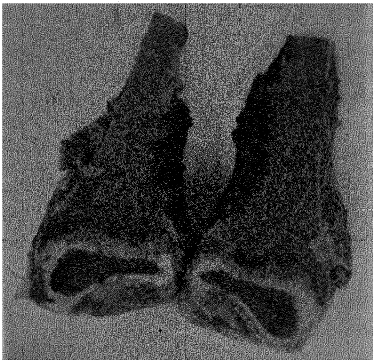

FIG. 19. The lower end of the radius from a case of renal rickets.

XEROPHTHALMIA

This condition is due to a deficiency of vitamin A. In this country it is uncommon, and when it occurs it is generally associated with rickets, but in some parts of the world, notably China,[1] xerophthalmia in infants is a serious and not uncommon condition, and may occur alone, or in company with rickets, or with other deficiency diseases such as beri-beri.

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[1] Graham, G., and Oakley, W. G., *Arch. Dis. Child.*, 1938, **13**, 1.

The essential lesion consists of a gradually increasing epithelial hypertrophy and opacity of the cornea, which may progress to ulceration and destruction. White patches also appear on the palpebral and ocular conjunctiva, and there may be severe conjunctivitis. An important early symptom is night-blindness —in children who are old enough to complain of it.

Treatment consists of supplying vitamin A by giving halibut-liver oil, which is about a hundred times richer in this vitamin than cod-liver oil. Five minims of halibut-liver oil should be given daily. The diet should also include fresh unboiled milk. If rickets is present it should not be treated by exposures to ultra-violet light unless the eyes are carefully protected.

INFANTILE SCURVY

For many years infantile scurvy and rickets were confused, scurvy being known as "acute rickets". The two conditions were separated by Cheadle in 1878, and a few years later Barlow fully described the morbid anatomy of infantile scurvy, but for some years confusion persisted in the use of the term "scurvy-rickets", owing to the frequent co-existence of both scurvy and rickets in the same child.

Etiology. *Diet.* It has been known for many years that the development of scurvy is associated with the lack of some substance in the diet, and that it can be cured by giving fresh fruit juice. The curative substance, which is present in considerable amount in lemon juice, is known as vitamin C. Orange juice is also a potent source of the vitamin, and the inclusion of orange juice in the diet of infants is now widely practised in order to prevent the disease. Grape-juice, the juice of swedes, and potato are also rich in vitamin C, as indeed are most fresh fruits and vegetables. Fresh tomato juice is almost as rich a source as orange juice, although when tomatoes are tinned and stored they lose much of their anti-scorbutic value.

The rarity of scurvy in breast-fed infants indicates that breast milk is a source of vitamin C, the mother obtaining it from her diet and transmitting it in her milk. Fresh unboiled cow's milk also contains the anti-scorbutic vitamin, but this is largely destroyed if the milk is boiled or pasteurised, and also in the processes of drying and condensing. When it is remembered that the great majority of infants in this country who are artificially fed are reared on cow's milk which has been submitted to one or other of these processes, the importance of adding fresh fruit juice to their diet becomes obvious.

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[1] Weech, A. A., *Amer. Jour. Dis. Child.*, 1930, **39**, 1153.

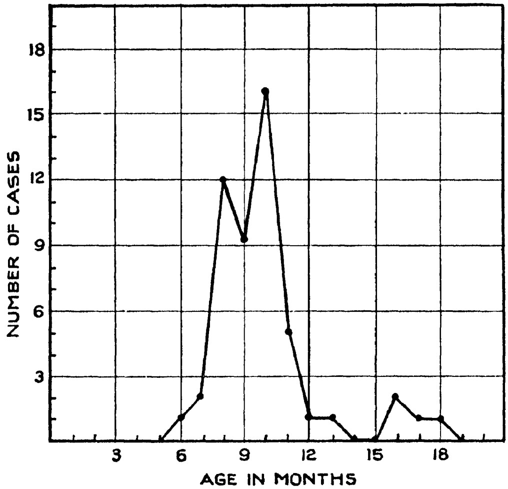

FIG. 20. Age incidence of fifty consecutive cases of infantile scurvy. (Great Ormond Street.)

The composition of vitamin C has recently been discovered, and its chemical synthesis carried out. It is known as *Ascorbic Acid*, or *Cevitamic Acid.*

Age. Infantile scurvy usually begins at about the ninth month, although the disease may arise at any time between six and eighteen months. It is very rare before six months. The age incidence of 50 consecutive cases is shown in the accompanying chart; none of the children had been fed at the breast within six months of the development of scurvy; the diet of nine of them had consisted of fresh milk sterilised by boiling or pasteurising, the remainder had been brought up on patent foods.

Symptoms. The onset is as a rule gradual, the early symptoms consisting of fretfulness and increasing paleness. These may, however, be overlooked, and then the first complaint is likely to be that the baby has lost the use of his limbs—generally the legs—and that he cries whenever they are touched. This is often first noticed when the legs are being dried at bath time. The general tenderness and the fear of being touched become evident as soon as the infant begins to be undressed, and his crying is aggravated when the limbs are handled during examination. Occasionally the onset is more abrupt, tenderness of the legs being noticed one morning, although only the day before the baby had seemed in good health.

The symptoms are mainly the result of hæmorrhages under the periosteum of the long bones, into the gums, and occasionally into the viscera. The position of the child is often characteristic, the legs are drawn up in a frog-like attitude with the thighs flexed and abducted and the knees flexed. The legs may appear to be paralysed, although this is actually not the case, for gentle stimulation of the sole of the foot will show that the infant can move all the joints of the limb. The tendon reflexes are also present, although when the diagnosis is already obvious it is a pity to give the child needless pain by attempting to elicit them.

The hæmorrhage under the periosteum is sometimes extensive enough to be visible as a diffuse swelling above or below the knee. The muscles may also be infiltrated with blood, and in severe cases the limbs may be œdematous. Palpation will confirm the presence of the subperiosteal hæmorrhages. In the legs the hæmorrhages are usually situated at the lower end of the femur or tibia, and in the arms at the distal end of the radius. Occasionally hæmorrhage takes place between the diaphysis and epiphysis, and the epiphysis may then become separated from the shaft of the bone.

Hæmorrhage into the gums is another common symptom. It is present only if the teeth have erupted, and usually occurs at the base of the incisor teeth in both upper and lower jaws. The gum may merely show a narrow line of purple discoloration, or there may be some swelling as well, but in severe cases the gums swell up into large purple fleshy masses which bleed as soon as they are touched, and the teeth may become loose or may even fall out. Occasionally hæmorrhages occur on the hard palate. Bleeding may also take place within the orbit, giving rise to proptosis, with swelling and bruising of the eyelids. One or both eyes may be affected.

Examination of the chest practically always shows scorbutic beading of the ribs. This is an early sign, and is present in over three quarters of the cases. The beading is produced by a backward displacement of the sternum and costal cartilages, as though these had been pushed into the chest, and this is the sensation conveyed when the costo-chondral junctions are palpated. The beading is sometimes due to hæmorrhage at the junction of the rib and cartilage, allowing a separation and backward displacement of the cartilage, but it is doubtful whether this is the whole explanation, for the beads are not as a rule tender, and this would certainly be expected in the presence of hæmorrhage. It is important to distinguish the beads of scurvy from those of rickets. In the latter the bead is a trumpet-shaped swelling of the end of the rib without backward displacement of the cartilage, and the sternum, instead of being depressed into the chest, often protrudes as a " pigeon breast ". Under treatment, the beads of scurvy begin to disappear within a few weeks, and have usually gone entirely after about three months.

Hæmaturia is another early and important symptom. Occasionally the urine is bright with blood, but this is unusual; much more often red corpuscles are only present in microscopic amount, and to the naked eye the urine seems normal. If scurvy is suspected, microscopical examination of the urine for blood is a useful means of confirming the diagnosis. The passage of blood in the stools is much less common, and when it occurs the blood is in small amount, and its passage is not accompanied by abdominal pain or tenderness. Cutaneous and visceral hæmorrhages, other than those mentioned, are rare.

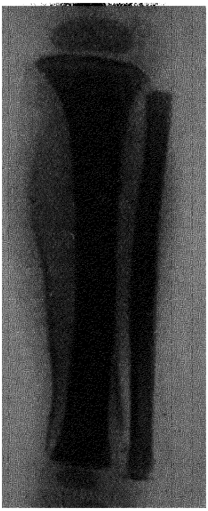

FIG. 21. X-ray of scurvy in a child one year old, showing density of the epiphyseal lines, and a shell of new bone outlining a hæmorrhage along the shaft of the left tibia. (By courtesy of Dr. Shires.)

Mention has already been made of pallor as an early sign of scurvy. Anæmia is almost always present, and is often severe enough to give the infant a waxy complexion. A blood count shows a reduction in both red cells and hæmoglobin, but especially the latter, the colour index being low. That the anæmia is due to the lack of vitamin C rather than to the amount of blood lost in the hæmorrhages is indicated by the fact that no matter what treatment is adopted the blood picture will not improve until the vitamin is restored to the diet.

So long as scurvy is present the process of growth ceases, and this gives rise to a characteristic X-ray picture, for calcium salts continue to be deposited at the epiphyseal lines, causing a narrow zone of dense calcification to appear at the ends of the long bones—the "white line" of scurvy. The appearance is very different from that of rickets, since the bone end is straight and regular instead of being widened, hollowed out, and irregular, but it must be remembered that the picture may be complicated by the co-existence of both diseases. The X-ray film in scurvy may also show separation of the periosteum from the shaft of the bone; and when the subperiosteal hæmorrhage has been present for two or three weeks the raised periosteum begins to form a layer of fresh bone which can be seen as a thin shell outlining the hæmorrhage. Eventually the hæmorrhage is slowly absorbed and the bone resumes its normal shape.

Diagnosis. The diagnosis is seldom difficult when there is a clear history that an infant, during the second half of the first year, has developed tenderness of the limbs. In the early stages the presence of scorbutic beading of the ribs is of great assistance, as may be also a microscopical examination of the urine for red blood cells, while if any teeth have erupted there may be bleeding into the gums.

Scurvy should not be confused with acute osteomyelitis, for although the latter gives rise to much pain and tenderness of a limb with swelling over the affected bone, the temperature is usually high (104° F. or higher), and the child is more acutely ill; generally the inflammation is confined to one bone, and furthermore, confirmatory signs of scurvy are lacking. Scurvy has also been mistaken for sarcoma when the swelling affected one limb only, but an examination for other evidence of scurvy should prevent this error. The age incidence distinguishes scurvy from syphilitic epiphysitis, which occurs under six months of age, and is usually accompanied by other evidence of congenital syphilis. The age should also prevent scurvy being mistaken for acute rheumatism, which is quite exceptional before the age of three years. Acute anterior poliomyelitis may sometimes be confused with scurvy, for in both conditions the limb is held immobile, and in the early stages of poliomyelitis the paralysed limb may be acutely tender. There is, however, no swelling of the limb in poliomyelitis, while a careful examination will show that the scorbutic limb is not really paralysed nor are the tendon reflexes lost. Scorbutic bleeding into the gums must not be mistaken for teething, for teething does not give rise to hæmorrhage, at least not more than the thinnest red streak at the point of eruption just when the tooth is appearing, while the purple discoloration of the gums in scurvy occurs at the base of teeth which have already been cut.

Prognosis. The symptoms disappear with remarkable rapidity as soon as vitamin C in adequate amounts is added to the diet. Within two or three days pain and tenderness pass off, and the hæmorrhages into the gums are generally absorbed within a week. Small subperiosteal effusions also absorb quickly, although the larger ones may take two or three months before completely disappearing. Microscopical amounts of blood cease to appear in the urine after a week or so.

A fatal outcome is likely in infants who are severely wasted, or may come about because of some intercurrent infection such as pyelitis or diarrhœa, to which scorbutic infants are particularly susceptible. The co-existence of rickets also affects the prognosis adversely.

Treatment. Great gentleness in nursing is necessary in order to prevent any unnecessary suffering. The cot should be lined with cotton wool, and the room must be kept sufficiently warm to allow the weight of the bedclothes to be raised off the child by putting a cradle in the cot. All movements of the infant are painful, and therefore he should be disturbed as little as possible, not being picked up and fondled in the hope of pacifying him The ordinary daily bath should also be omitted until all tenderness has gone

The essence of treatment lies in giving an adequate amount of anti-scorbutic vitamin. If the infant is being brought up on a patent food or dried milk, he should be taken off this and be given fresh unboiled cow's milk As the milk is to be given unboiled, any risk of infection should be reduced as far as possible by ordering tuberculin-tested milk Fresh fruit juice must be given daily, the juice of a whole orange being given during the twenty-four hours, or at least an equivalent amount of grape or tomato juice. Decitrated[1] lemon juice may also be used if it is diluted and well sweetened.

Potato has a well-deserved reputation in the treatment of infantile scurvy. It is given in the form of potato cream, which is prepared by baking a potato in its jacket and then scraping the soft floury part from just under the skin and beating this up with fresh milk. Roughly a teaspoonful of potato should be mixed with an ounce of milk. Two ounces of potato cream should be given daily until all the symptoms have cleared up. Raw meat juice has also been recommended, but it is doubtful whether it possesses much anti-scorbutic value. Unboiled milk, fresh fruit juice, and potato cream can be relied upon to bring about a rapid improvement in two or three days.

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[1] Lemon juice may be decitrated by saturation with calcium carbonate, when the citric acid is precipitated as calcium citrate. Decitrated lemon juice must be freshly prepared.

The pure vitamin in the form of ascorbic acid is now on the market. It is put up in tablets containing 5, 25, and 50 mgms., and at least 50 mgms. should be given in the day. There seems to be no danger from over-dosage, for any excess is excreted in the urine. Pure ascorbic acid is particularly useful when the digestion is so disordered that the infant is unable to tolerate the usual dietetic measures. In a severe case an initial dose of 50 mgms. may be given intravenously.

Drugs play practically no part in the treatment, but small doses of chloral hydrate (gr. 2 ter die) may be used for a day or two to soothe the infant and lessen his pain.

Prevention. The freedom from scurvy of the breast-fed infant is one good reason, among many, for encouraging a mother to suckle her infant. With regard to cow's milk, the small amount of anti-scorbutic vitamin in it is largely destroyed when the milk is boiled or pasteurised, although, for the reasons given in an earlier chapter, sterilisation of fresh milk is highly desirable. Dried or condensed milk and the various patent cereal foods are also devoid of vitamin C, so that steps to prevent scurvy must be taken in all artificially reared infants.

Vitamin C is added to the infant's diet usually in the form of fresh fruit juice. Although lemon juice is richer in anti-scorbutic vitamin than other fruit juices, its high acidity makes it difficult to give to infants, but orange juice is also rich in the vitamin and is generally tolerated very well. It should be given daily from the age of two months, beginning with a teaspoonful and gradually increasing until the juice of a whole orange is being taken at six months. The juice should be diluted with a little warm water and sweetened. Occasionally orange juice causes some looseness of the bowels, and there are infants who so dislike its taste that they absolutely refuse it. The juice of grapes may then be used instead, although it is not so rich a source of vitamin C as is orange juice. The juice of *fresh* tomatoes may also be used, for although it contains slightly less of the vitamin than orange juice it is stronger than grape juice. Seeing that fruit juice loses much of its anti-scorbutic property if stored for any length of time, the various proprietary preparations of bottled fruit juice cannot be recommended. The wartime use of black currant purée, rose hip syrup, and ascorbic acid tablets, is outlined in the footnote on p. 71.

It not infrequently happens that when a mother is advised to give her infant orange juice she thinks that its purpose is to act as a laxative, and then if the bowels are working satisfactorily the fruit juice is withheld. The reason for giving fruit juice should be properly explained to her.

PELLAGRA

Although pellagra is a rare disease in this country, its occurrence in children is reported from time to time. Box has recorded a child in whom the symptoms first appeared at the early age of twenty months. Recent investigations have shown that the condition is due to lack of the nicotinic acid fraction of the vitamin B complex.

The disease is characterised by erythema and thickening of certain areas of the skin, together with progressive degeneration of the nervous system. The symptoms are aggravated each spring and summer, with remissions during the winter.

The onset is generally insidious, the child becoming dull and irritable. There may be a complaint of headache and giddiness, the gait becomes unsteady, and tremors or sudden spasmodic movements of the extremities may develop. Paræsthesiæ or areas of anæsthesia may occur in the limbs. Digestive disturbances, with a sore tongue, gingivitis, vomiting, and diarrhœa, may arise.

The skin lesions affect those parts exposed to the sun, namely the face, neck, hands, and in children perhaps the legs, beginning as a severe sunburn with swelling and tenderness. Later the skin desquamates and becomes thick and rough, while the initial erythema remains as a brown staining.

The course may extend over five or six years, with exacerbations each spring. Gradually the child becomes emaciated, demented, and paralysed, and death results from exhaustion.

Treatment. During the early stages, improvement may be brought about by feeding on a high protein diet containing meat, eggs, wheat and casein, together with a daily dose of 15 gms. of yeast. The normally exposed parts of the skin should be carefully protected from the sun. When the disease is seen for the first time in an advanced state, treatment has little effect.

Excellent results have been reported to follow the oral administration of nicotinic acid. In a series of 73 patients, including 36 children, treated by Spies[1] the dose was from 100 to 200 mgms. daily.

BERI-BERI (POLYNEURITIS ENDEMICA)

Beri-beri is a disease of tropical and subtropical countries, and is hardly ever met with in this country. The symptoms are due to a multiple peripheral neuritis arising from a deficiency in the diet of vitamin B_1 (aneurin : thiamin). This vitamin is contained in the protein or germ of cereals such as wheat and rice.

During childhood beri-beri is commonest under two years of age, particularly in the breast-fed infants of mothers suffering from the disease. In its most virulent form, infants of two or three months old are seized with a series of convulsions terminating fatally ; in the more protracted cases there is progressive weakness and emaciation accompanied by restlessness, breathlessness, and aphonia. A generalised œdema of the tissues may appear. Blood examination shows a leucopenia with an absence of small lymphocytes. Death, which may be sudden, results from cardiac dilatation and failure.

Treatment. This is mainly dietetic. The vitamin must be replaced in the diet by incorporating such foods as milk, eggs, wheat flour or barley. White rice should not be allowed. If the infant is still at the breast, the mother's diet must be improved along the lines indicated above. There is a plentiful supply of the vitamin in yeast, a teaspoonful of which should be given daily ; or marmite, which is an extract prepared from yeast, may be given in doses of 1 gm. daily. The pure vitamin may also be obtained in tablet form, and in ampoules for intramuscular injection.

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[1] Spies, T. D., Bean, W. B., and Stone, R. E., *J.A.M.A.*, 1938, **110**, 645.

CHAPTER VIII

DISORDERS OF METABOLISM

ACIDOSIS AND ALKALOSIS

THE balance between the acids and bases of the blood and body fluids is in health maintained at a remarkably constant level, which is just on the alkaline side of neutrality. The term *acidosis* implies that the normal acid-base balance has been upset, and that there is an excess of acids, and similarly a state of *alkalosis* is present when the amount of base is allowed to accumulate, but in neither case is there much actual alteration in the reaction of the blood. This is because of the presence in the blood of " buffer " substances—chiefly proteins, carbon dioxide, bicarbonates and phosphates—which have the property of taking up excess of acids or alkalies without appreciably altering the reaction of the fluid containing them. Any but the slightest changes in the blood reaction would be incompatible with life. The various methods of estimating these changes, such as the estimation of the alkaline reserve and the CO_2-combining power of the blood, need not concern us here. Both acidosis and alkalosis may be brought about in a variety of ways and may be met with in the course of several diseases, but they do not occur as isolated entities and therefore must not be allowed to rank as individual diseases.

Much confusion has hitherto resulted because " ketosis " and " acidosis " have been used as synonymous terms, acidosis being allowed to cover the presence of ketone bodies (acetone, aceto-acetic acid, β-hydroxybutyric acid) in the blood and urine. These substances are formed from the incomplete oxidation of fat, which takes place when the body is short of, or is unable to utilise, carbohydrate. The formation of these substances is more correctly indicated by the term *ketosis*, their presence in the blood and urine being spoken of as *ketonæmia* and *ketonuria* respectively. It is true that acidosis may sometimes be brought about by an excessive production of ketone bodies, but they may also be found when there is no alteration of the acid-base balance, and indeed may be present in company with an actual alkalosis. It should therefore be apparent that the terms acidosis and ketosis refer to different conditions, and the difference in their meanings must be borne in mind.

Acidosis

There are three ways in which acidosis may be brought about. The normal excretion of acids may break down, allowing acid substances to accumulate ; or an excessive loss of bases may take place ; or acid substances not normally present may be formed in sufficient amount to provoke an acidosis. A failure in the excretion of acids is met with in certain severe forms of nephritis, both acute and chronic, and particularly in association with uræmia. The symptoms attributable to the acidosis are dyspnœa, with a hissing or even asthmatic type of breathing, and drowsiness. This type of acidosis should be treated by supplying the body with extra alkali in the form of sodium bicarbonate. Acidosis brought about by the depletion of the body's store of bases occurs during a sharp attack of diarrhœa, the bases being lost in the stools, although the likelihood of acidosis may to some extent be countered if the diarrhœa is accompanied by vomiting, for the loss of hydrochloric acid in the vomit will help to compensate for the loss of bases in the stools. Should the symptoms of acidosis accompany an attack of diarrhœa, they should be treated by replenishing the loss of fluid and by giving sodium bicarbonate.

Acidosis of the third variety—due to the formation of acid substances not normally present—is generally the result of the accumulation of ketone bodies. This is seen most convincingly in diabetes, and accounts for the air-hunger which may be so prominent a symptom of diabetic coma. The correct treatment of this variety of acidosis lies in enabling the body to utilise its sugar in order to ensure a complete oxidation of fats, and this will be accomplished by giving insulin and glucose.

Alkalosis

Alkalosis may be brought about in two ways. In the first place it may be due to excessive loss of acid, such as occurs in repeated vomiting. Thus it is met with in hypertrophic pyloric stenosis, in high intestinal obstruction (duodenal atresia, volvulus, etc.), and in peritonitis. Experimentally, an excessive loss of CO_2 in the breath by forced over-breathing may also lead to alkalosis, and clinically this may be met with in the tachypnœic attacks which are sometimes a sequela of epidemic encephalitis. Secondly alkalosis may be produced by giving large amounts of alkali in the treatment of disease, and thus may come about during the treatment of pyelitis and nephritis.

The symptoms of alkalosis are less definite than those of acidosis, and comprise drowsiness, shallow breathing, muscular weakness, headache, and mental confusion. In infants with pyloric stenosis, alkalosis may show itself by unusual lethargy and by difficulty in getting the infant to evince any desire for, or interest in, its feeds. The more severe degrees of alkalosis are likely to be accompanied by tetany, and may lead to coma.

The treatment must depend largely on the cause. Thus a high intestinal obstruction calls for prompt surgical relief, while alkalosis from excessive doses of alkali will quickly disappear when the drugs are stopped. The alkalosis of hypertrophic pyloric stenosis should be dealt with by washing out the stomach with normal saline—instead of sodium bicarbonate which is usually used for gastric lavage—and by infusions of normal saline, the object being to replace the chlorine ions lost in the vomit.

Ketosis

A ketosis of severe degree, such as may occur in diabetes mellitus, will give rise to acidosis, but there are many other conditions in childhood in which ketones are formed in detectable amounts and yet may not affect the acid-base balance. There is no doubt that children are much more liable to the formation of ketone bodies than are adults, because their store of carbohydrate in the liver is smaller and so is more quickly exhausted. The immediate result is that the metabolism of fat (either the fat of the diet or endogenous fat) is incompletely carried out, and this leads to the formation of β-hydroxybutyric acid, acetoacetic acid, and acetone. A few hours of starvation is often sufficient to deplete a child's carbohydrate reserve and lead to ketosis, and thus it is met with in gastro-intestinal disorders, and after pre-operative starvation. It also occurs if the metabolic rate is rapidly raised, such as during sharp bouts of fever (tonsillitis, pneumonia, pyelitis, etc.).

Cyclic Vomiting

True cyclic vomiting is not a common condition. It is characterised by attacks of severe vomiting associated with much prostration and accompanied by heavy ketosis, the attacks recurring at remarkably regular intervals. There is no doubt that similar symptoms frequently accompany acute feverish illnesses in children, expecially infections of the nasopharynx, and as these may be recurrent they are apt to be mistaken for cyclic vomiting, but they do not occur with the regular periodicity which is so typical of the latter condition.

Cyclic vomiting is equally distributed between the sexes, and is essentially a condition of childhood. The first attack usually develops before the second birthday, and there is a natural tendency for the attacks to disappear as adolescence approaches, although they are sometimes replaced at that age by the symptoms of migraine. Occasionally several children of a family are subject to these attacks, and the highly-strung type of child, spare of build and full of restless nervous energy, is particularly likely to be affected. That the period between attacks is remarkably constant has already been commented upon, but the interval varies with each individual child from perhaps once in three weeks to once every three months. The condition is more often met with among children of the better classes, probably because of the tendency to feed them up in the interval between attacks with fatty foods, cream, and emulsions of one sort and another, thereby laying the foundation for the next attack.

Symptoms. The onset of an attack may be sudden, beginning with vomiting, but more usually premonitory warnings occur for a day or two and consist of irritability or drowsiness, anorexia, a furred tongue, and tainted breath. Vomiting follows, and is soon incessant, even water being returned. The tongue becomes dry and the lips parched, the child is restless and thirsty, and there may at first be much headache, to be soon replaced by drowsiness. As a rule the bowels are constipated, and the motions become pale. Fever is usual, and may reach 103° F. or higher. Complaint of abdominal pain is usual, and there may be some tenderness over the epigastrium, but it is characteristic that these symptoms do not appear until some little time after the vomiting, and are probably caused by the straining of the abdominal wall owing to the incessant retching. The urine becomes scanty and highly coloured and may contain albumin. Ketone bodies can be detected in the urine in large quantities, and their elimination in the breath may give it a characteristic sweetish odour.

As the attack progresses drowsiness may pass on to coma, the eyes become sunken, and the abdomen becomes hollowed out. The appearance of the child may be most alarming. Slight retraction of the head and a positive Kernig's sign may lead to a mistaken diagnosis of tuberculous meningitis, especially as in the most severe cases there may be convulsions.

As a rule the attack abates after three or four days, although it may drag on for a week. Naturally enough the child is exhausted at the end of the attack and may have lost several pounds in weight, but a fatal issue is fortunately very rare. Once the attack is over the child usually makes a rapid return to a normal state of health.

Pathology. Very little is known about the pathology of cyclic vomiting. In the few cases that have come to autopsy the most noticeable change has been the severe degree of fatty degeneration in the liver, and to a less extent in the kidneys and heart, but these changes are found commonly enough in children who have died from other conditions. It is clear that each attack is accompanied by severe metabolic disturbances, and it seems likely that the liver function of these children is particularly unstable, and that their hepatic glycogen store is capable of a too rapid depletion. Between attacks the level of the blood sugar is well up to normal. It seems probable that the ketosis is the result, and not the cause, of the vomiting, for children who are known to be subject to these attacks have been fed intentionally on a ketogenic diet until ketone bodies have appeared in the urine, but without provoking an attack.

Diagnosis. The diagnosis can only be reached after a thorough examination has been made to exclude other conditions in which fever, vomiting, and ketosis may be associated, expecially infective states such as sore throat, otitis media, and pyelitis. A history of similar attacks having occurred previously at regular intervals must always be suggestive, provided it is borne in mind that infective conditions such as those enumerated above may also recur—but at irregular intervals.

A clear history that vomiting has occurred some hours before the onset of abdominal pain is of value in distinguishing cyclical vomiting from the various causes of acute intestinal obstruction, especially appendicitis. If the tenderness can be localised it will usually be found over the upper abdomen, which is also unlike appendicitis, but even so there may be the greatest difficulty in separating cyclic vomiting from such conditions as recurrent appendicitis, intussusception, and partial volvulus. The retraction of the abdomen, which becomes more noticeable as the attack becomes more severe, is also unlike obstructive conditions.

Treatment. During an attack the child will, of course, be in bed. As much as possible of such simple fluids as water or sweetened lemonade should be given, and if vomiting prevents fluid by mouth being retained, the stomach should be washed out with a solution of sodium bicarbonate (a drachm to a pint). No attempt should be made to give milk or other foods. Sugar by mouth in the form of glucose is necessary to combat the ketosis and there is advantage in giving sodium bicarbonate as well. A teaspoonful of the latter with two teaspoonfuls of glucose should be dissolved in a tumbler of water, and given in sips, three or four tumblerfuls being taken in the day. When vomiting is so persistent that this cannot be carried out the rectum should be washed out and then a drip enema of 10 per cent. glucose solution, containing a drachm of sodium bicarbonate to the pint, should be given. If dehydration becomes severe a subcutaneous infusion of 2½ per cent. glucose in normal saline may be given, or a 10 per cent. solution of glucose in normal saline may be injected intravenously. As soon as vomiting desists extra carbohydrate should be supplied in the form of toast or rusks with honey and stewed fruit.

The prevention of further attacks is largely a dietetic matter. The principle should be to give a diet rich in carbohydrate but low in fat. The fat of cow's milk is particularly badly tolerated, and therefore when milk is given in any form it should first of all be skimmed. Cream and cheese must be forbidden, and only a thin scraping of butter should be allowed. Eggs should be limited to three a week, and chocolate and cocoa should not be given, although boiled sweets, which consist almost entirely of sugar and water, are harmless. The bulk of the diet will be made up from bread, potato, vegetables, fruit, lean meat, fish, jams, and cereal puddings made with skimmed milk. In addition, a teaspoonful of glucose may usefully be incorporated in the drinks at mealtimes.

Attention should be paid to the regular action of the bowels, magnesia being given when necessary. There is often a history that excitement and undue fatigue have played some part in promoting attacks, and these should therefore be avoided.

Conditions Allied to Cyclic Vomiting

The recurrent breakdown of fat metabolism, which is the underlying factor in producing cyclic vomiting, may find other clinical expressions, for instance, recurrent bouts of diarrhœa with the passage of loose pale offensive motions may occur—this was so in a little boy of five years of age, whose parents took pride in his daily ration of a pint and a half of rich Jersey milk. Occasionally cyclic attacks of asthma occur, and periodic bouts of migraine are sometimes met with in older children. Rarely epileptic seizures may also show a regular periodicity. In these various conditions the one feature common to them all is the regular interval between the attacks, so much so that the parents may be able to tell almost to the day when the next attack is due. Inquiry will generally show that an attempt is being made to build up these children between their attacks by giving a generous and rich diet, and a good response to treatment is not likely to come about until the diet has been modified in the direction of curtailing the fats and increasing the intake of sugars.

DIABETES MELLITUS

Diabetes mellitus is uncommon before the middle years of childhood. Lawrence and McCance [1] have recorded an instance of severe diabetes with gangrene in an infant only eighteen days old, and were able to collect from the literature records of 26 diabetic children under one year of age. Although in adults males are more often affected than females, the sex incidence in children is equally distributed. In roughly a quarter of the cases there is a history of diabetes in the parents or other members of the family, but an hereditary influence does not tend to make the disease more severe.

Symptoms. The onset in children is usually more acute than in adults, and not infrequently the symptoms develop so rapidly that only a week or two elapses before medical advice is sought. There is sometimes a history of one of the acute exanthemata having immediately preceded the onset.

The first symptoms are generally thirst and polyuria, and as in children the latter may give rise to enuresis the first complaint may be of bed-wetting. Listlessness, rapid fatigue, muscular weakness, and loss of weight soon follow, while the appetite may become voracious. The skin becomes dry and sallow, while the lips in contrast may seem unusually red, the tendon reflexes become depressed, and the breath may smell of acetone. In girls itching of the vulva may be an early complaint, and septic infections of the skin are likely. The urine is pale, with a high specific gravity (1,030 to 1,040), and contains sugar and almost always ketone bodies as well. The level of the fasting blood sugar is raised (normal in children, 60 to 120 mgm. per 100 c.c.).

Complications. The most important is coma. The onset is usually marked by repeated vomiting and often abdominal pain, followed by increasing drowsiness, deeper and quicker respirations (air-hunger), and finally coma. The blood pressure falls, and there is a detectable fall in the tension of the eye-ball. The breath smells strongly of acetone, and the urine contains both sugar and ketone bodies. The onset with vomiting and abdominal pain may at first suggest some acute surgical condition, but mistakes will be avoided by examining the urine.

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[1] *Arch. Dis. Child.*, 1931, **6**, 343.

Of other complications, attacks of fever due to mild infections of the skin or throat, which incidentally are common enough in all children, have for the diabetic child an added importance because they call for an extra combustion of carbohydrate. So it comes about that such simple conditions as a common cold or a sore throat may precipitate a diabetic child into coma, or, if the child is already under treatment, may call for a temporary increase in the dose of insulin. In the same way the acute infectious fevers, which are chiefly the concern of children, are also likely to aggravate the diabetic condition.

Retinitis, gangrene, tuberculosis—complications of importance in adults—are all rare in children.

Diagnosis. The diagnosis in childhood seldom presents difficulty because, in addition to glycosuria, the other symptoms such as thirst, polyuria, loss of weight and so on are so acute. Reducing substances in the urine other than glucose, such as lactose or pentose, are very rare, but may be distinguished by the characters of their osazone crystals, and by a glucose tolerance test.

It is important to distinguish between diabetes mellitus and renal glycosuria—a condition in which sugar passes from the blood into the urine because the renal threshold for glucose is unusually low—for the latter is harmless and does not call for treatment in spite of small amounts of glucose repeatedly appearing in the urine. Normally, sugar does not appear in the urine until the blood sugar has reached a level of 180 mgm. per 100 c.c., and therefore a simultaneous study of the blood-sugar level and the appearance of glucose in the urine will at once decide whether the glycosuria is due to a lowered renal threshold or to an abnormally high blood-sugar level as in diabetes mellitus.

Glycosuria may also appear in children as a temporary affair, sometimes from an over-indulgence in sugar, or during acute infections. It may also be present when there is an increased intracranial tension, for instance after injuries to the head, and is commonly to be found towards the end of tuberculous meningitis—at a stage when the evidence of meningitis is so unmistakable that confusion with diabetes is hardly likely to arise. It may occasionally mislead at the onset of meningococcal meningitis. In general terms a coma which is accompanied by glycosuria and ketonuria should be regarded as diabetic unless there is very good evidence to the contrary. On the other hand if a child who is under treatment with insulin passes into coma, the possibility of it being due to hypoglycæmia must be borne in mind (see later).

Prognosis. Before the discovery of insulin the outlook for the diabetic child was practically hopeless, for even with the most careful dieting, life could seldom be prolonged for more than two or three years, and often the course was only a few months. The prognosis has entirely altered since the advent of insulin, and at the present day if the diagnosis is made reasonably early and the dietetic and insulin treatment is accurately carried out, a child may be expected not only to survive but to thrive and develop at a normal rate. Whether complete recovery, in the strict sense of the word, can come about is still a matter of doubt, but it is certain that a healthy and active existence can be maintained over many years, and this in spite of the fact that children are more difficult to control by reason of their greater liability to infections, their sudden bursts of vigorous exercise, and their more capricious appetite, which may lead to insulin overdosage. The manner in which diabetic children will stick to their diet and submit to insulin injections over years is truly astounding.

Treatment. Insulin has entirely changed the treatment of the child diabetic, and starvation days and minimal diets are things of the past. The position may be summarised by saying that the child is put on to a diet which will easily cover the physiological requirements ; should this lead to glycosuria or to a raised blood-sugar level, insulin is given In practice almost all diabetic children require insulin.

A satisfactory diet for a child must of course be sufficient to allow for growth, and this calls for a higher proportion of protein than in an adult's diet. Then, too, the younger the child the higher must be the number of calories per kilogram of body-weight because growth is most active in young children and also the heat loss is relatively greater owing to the relatively larger skin surface. The proportion of carbohydrate to fat must also be generous enough to minimise the likelihood of ketosis, to which young children are particularly prone.

Construction of the Diet.[1] The construction of a diet for a diabetic child has recently become much simplified owing to the increased proportion of carbohydrate which insulin therapy permits. Careful measurements of protein and fat foods are no longer necessary, all that is required is that the amount of carbohydrate shall be sufficient to enable average helpings of protein and fat foods to be taken. The amount of carbohydrate that should be allowed is set out in the following table, and this amount must be correctly measured.

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[1] With acknowledgments to R. D. Lawrence's "A Simple Diabetic Diet (Unweighed)." Published by H. K. Lewis & Co. Ltd.

Age in years.	Minimum daily allowance of carbohydrate.
Up to four years	100 gm.
Four to eight years	120 gm.
Nine to twelve years	150 gm.

The type of diet will, of course, vary according to the age, for instance, during the first year it must be composed mainly of milk, and until four years the diet will not depart in any great measure from that given to an ordinary child, indeed, throughout childhood a modern diabetic diet allows of a tolerably normal fare being taken.

Turning now to actual food-stuffs, the following are composed of proteins and fats, and may be taken in average helpings :—

Beef
Veal
Mutton
Poultry
Pork
Ham
Bacon
Suet
Liver
Kidney
Eggs
Fish
Cheese
Butter
Cream
Dripping

The carbohydrate foods may be conveniently divided into the following three groups :—

I. *Allowed in Unlimited Amount—*

Cabbage
Cauliflower
Greens
Spinach
Seakale
French beans
Scarlet runners
Marrow
Tomato
Celery
Lettuce
Cress
Radishes
Cucumber

Gooseberries
Rhubarb
Currants (black or red)
stewed.

II. *Three Tablespoonfuls of one of these contains* 5 *gm. of Carbohydrate—*

Brussels sprouts
Carrots
Turnips
Onions
Leeks
Artichokes

Apples
Apricots
Blackberries
Cherries
Damsons
Greengages
Pears
Plums
stewed.

Strawberries
Raspberries
1 orange or
1 peach or 1 plum or small apple
Half grape fruit
Melon, 1 slice
fresh.

III. *To be measured accurately. Each portion contains* 10 *gm. Carbohydrate*—

Bread,* ⅔ oz. (a piece measuring 4 × 3 × ¼ inch).
Potato (size of a hen's egg).
Milk, 7 oz. (teacup).
Rice, tapioca, oatmeal (1 dessertspoonful).
Two oranges or 2 peaches or 2 plums or ¼ lb. apple or pear.
Benger's Food or Horlick's Malted Milk, 2 teaspoonfuls.
Sugar, 2 teaspoonfuls or 2 lumps.
Plain biscuits, ½ oz., such as 2 " breakfast " or 2 " petit beurre".
Crispbreads (Ryvita, Vitaweat), 2 sections.

* H. K. Lewis & Co. supply a metal measure for this portion of bread.

The following foods should not be taken because of their high content of carbohydrate.

Sugar, sweets, jams, flour, cakes, pastry, peas, beans, dried fruits, grapes, dates, figs, pineapple, porridge, rice, tapioca, sago, sausages.

Insulin. It has already been pointed out that the majority of child diabetics require insulin. Owing to the rapid fluctuations in the blood-sugar level in childhood it may not be possible to keep the urine free of sugar throughout the twenty-four hours, but the dose of insulin should be sufficient to ensure that the urine passed four and a half hours after an injection shall be sugar-free. The child should be made to pass urine three hours after insulin in order to get rid of urine which may contain sugar, and then a second specimen should be passed an hour and a half later. This second specimen must be free of sugar, and a test on these lines should be carried out every day. In a mild case a start may be made with 3 or 5 units once a day, but in more severe cases the commencing dose should be two injections daily of 5 units each, the injections being given half an hour before breakfast and tea. The diet should be so arranged that these are the two largest meals of the day. The dose is then increased by 5 units in the morning, then 5 units in the afternoon, and so on until the tests of the urine become satisfactory. The injections are given subcutaneously, the mother or some responsible person in the house being trained to do this. Older children can be trained to give their insulin themselves, but arrangements should always be made for each dose to be witnessed by someone else. Injections should be given in various sites, using the arms and thighs, in order to prevent any hardening of the tissues or local atrophy of the subcutaneous fat.

Zinc-Protamine-Insulin. The advantage of this preparation lies in its slow absorption and prolonged action, which lasts over twenty-four hours. The number of daily injections may thereby be reduced to one, given half an hour before breakfast. The amount required may be worked out by the following technique,[1] for which blood sugar estimations are not essential. The bladder is emptied each morning at 7 a.m., and again at 8 a.m., and a satisfactory and safe dose is one which gives no sugar, or only a trace, in the 8 a.m. specimen on two mornings out of three. If sugar is absent from the 8 a.m. specimen every morning the dose is too large, and there is a risk of hypoglycæmic attacks during the night, and it is for this reason that the preliminary investigations should be conducted while the child is under observation in a hospital or nursing home. The full effect of protamine-insulin can only be estimated after three days of treatment, and therefore this period should elapse in judging the efficacy of any particular dose. In childhood the dose is likely to be between 10 and 20 units. If the diabetic condition is so severe that a trace of sugar in the 8 a.m. specimen goes with considerable glycosuria during the day time, the injection of protamine-insulin should be combined with sufficient ordinary insulin to control the day time glycosuria. The two forms of insulin may be given together, ordinary insulin being drawn into the syringe before the protamine-insulin.

Hypoglycæmic attacks after protamine-insulin differ in no way from similar attacks after ordinary insulin, but owing to the prolonged action of protamine-insulin, treatment of an attack may have to be repeated once or twice. Zinc-protamine-insulin should not be used in the treatment of diabetic coma, as its action is too slow.

Treatment of Coma. The essential is to give insulin until the blood sugar has fallen to within normal limits, or until the urine has been rendered sugar-free. It is possible to deal with coma quite efficiently without resort to repeated estimations of the blood sugar, but in that case glucose should be given at the same time as the insulin, and it is of the utmost importance that the urine be repeatedly tested for sugar. The following scheme may be employed :—

(1) Give insulin twenty units every four hours, together with glucose 20 gm.

(2) Test urine with Benedict's solution *before each dose of insulin.* This is essential, and a catheter should be passed if necessary to obtain a specimen.

(3) When the blue copper solution is no longer completely

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[1] Lawrence, R. D., *Brit. Med. Jour.*, 1939, 1, 1077.

reduced, but turns green with a red deposit of copper, reduce the dose of insulin to ten units every four hours, continuing with glucose 20 gm.

(4) When the test remains blue, that is to say, the urine no longer contains sugar, give insulin five units every four hours with glucose 20 gm. until a diet can be resumed.

When the coma has been overcome, the correct amount of carbohydrate for the age should be given, balanced with insulin, but the fat and protein part of the diet must be added more slowly as the child's appetite for these foodstuffs returns.

Sodium bicarbonate in the treatment of coma has now fallen into disrepute, but a solution containing two teaspoonfuls to the pint may be used as a gastric lavage when vomiting is severe and persistent. A great loss of fluid from the body occurs during the pre-coma period, and it is important that this should be replaced. Normal saline may be given per rectum or subcutaneously, but if the child is dangerously collapsed intravenous saline should be administered by the continuous drip method (p. 100).

Hypoglycæmia

Hypoglycæmia is more likely to occur in child diabetics who are under treatment with insulin than in adults, because of their sudden outbursts of exercise and their vagaries of appetite and even refusal of food. On the other hand, the blood sugar may fall to much lower levels in children without giving rise to symptoms, as low, for example, as 40 or even 30 mgm. per 100 c.c. The symptoms are also likely to be less obvious in children. At the onset the child may be paler than usual, quieter, or fretful. This gives place to drowsiness, inco-ordination of movement, paralysis, and eventually coma. A fit at the onset is not unusual. Odd behaviour or unusual lethargy in a child who is receiving insulin should be enough to arouse one's suspicions. Hypoglycæmic attacks are most likely to occur about three to four hours after a dose of insulin.

The treatment is to supply the child with sugar. In a mild case one lump of sugar dissolved in water, weak tea, or fruit juice may be all that is required. The sugar should always be given in solution so that it can be rapidly absorbed. The dose should be repeated at fifteen-minute intervals until an improvement is obvious. Larger amounts of sugar are seldom required, but if the coma is deep enough to interfere with swallowing, the solution of sugar should be given through a stomach tube, or a small intravenous injection of glucose may be given.

INBORN ERRORS OF METABOLISM

This name was given by Garrod to a group of disorders, each member of which is characterised by an abnormality of chemical function, present at birth and persisting through life. With advances in our knowledge of the chemical processes at work in the body it is likely that fresh members will continue to be added to the group. There are certain features common to them all : they are very rare ; they tend to appear in several members of a family, and though the parents are unaffected they are often consanguineous, indicating that the fault of metabolism may be looked upon as a Mendelian recessive character. The symptoms produced by the perverted chemical function may or may not be present at birth, indeed in some instances, such as pentosuria, the subjects may be symptomless.

Albinism. This is due to a failure in the formation of the melanin group of pigments. The hair is bleached, the skin is unpigmented, the irides are colourless, the pupils appear pink owing to the red retinal reflex being visible, and nystagmus is usually present.

Cystinuria. The error here lies in an inability to break down the cystin fraction of the proteins of the diet, and cystin crystals are passed in the urine. Symptoms arise from the formation of cystin calculi. These are opaque to X-rays, and are creamy-white in colour, but gradually turn a greenish hue when preserved for some time.

Alkaptonuria. This is due to the excretion of homogentisic acid, derived from the amino-acid tyrosin. Although when the urine is first passed its colour is normal it quickly turns dark brown, and this is the main symptom. The dark urine stains the clothes, and as the condition dates from birth the staining may first be noticed in the baby's napkins. In later life the cartilages blacken (ochronosis), the ears appear bluish, and brown patches appear on the sclerotics.

Pentosuria. The two pentoses that may be excreted in the urine are arabinose and xylose. The condition is symptomless and harmless, but as the urine reduces Fehling's solution it must be distinguished from diabetes mellitus, either by means of Bial's test or by a blood-sugar curve following the ingestion of glucose.

Congenital Porphyrinuria. The chief feature of this condition consists of the passage of deep red urine, due to the presence of the iron-free pigment hæmatoporphyrin, which may be detected by spectroscopic examination. The teeth and the bones may be pink or brownish from the presence of the pigment, while porphyrin in the skin renders the subjects very sensitive to light. As a result, the skin of exposed parts readily blisters, giving rise to a bullous eruption—hydroa vacciniforme. The eruption heals by scarring, and this may gradually lead to unsightly deformities. The liver and spleen are usually enlarged.

The formation of hæmatoporphyrin cannot be stopped, but the skin lesions may be prevented by protecting the parts from sunlight, and by the use of a skin cream containing quinine which filters off the ultra-violet rays.

Congenital Steatorrhœa. This very rare condition is characterised by the passage in the stools of a liquid oil, which solidifies on cooling. Growth is not interfered with. There is no evidence of pancreatic insufficiency. The condition may be distinguished from cœliac disease by its familial incidence, an onset from birth, a lack of free fatty acid in the stools, and the fact that the fat in the stools is passed in a liquid form.

DISORDERS OF LIPOID METABOLISM

The lipoids in the blood consist of cholesterol, phosphatides, which are compounds of phosphoric acid with a fatty acid and a nitrogen-containing base, and cerebrosides, which are compounds of a carbohydrate with a fatty acid and a nitrogen-containing base. Normally the body is able to dispose of these substances, but under certain circumstances not at present understood a gradual accumulation of them takes place, until sooner or later life may be endangered. Thus a storage of cerebrosides takes place in Gaucher's disease, of phosphatides in Niemann-Pick's disease and amaurotic family idiocy, and of cholesterol in the Schüller-Christian syndrome.

Gaucher's Disease

The main feature of this condition, which was first described by Gaucher in 1882, is progressive enlargement of the spleen. Frequently more than one child in a family is affected, and it is more common in boys than girls.

Although fatal cases have been recorded during the second year of life, the onset is a gradual one and attention is seldom drawn to the enlarged spleen before the middle years of childhood. The spleen may become enormous, extending down below the umbilicus, and attracting attention by the distension of the abdomen. It is smooth and not tender, although there may be much perisplenitis, but on account of its size it is likely to cause discomfort or pain in the left half of the abdomen. The liver is also enlarged, although to a less extent than the spleen. The general health is surprisingly little affected, but there may be some secondary anæmia, or a faint icteric tinge to the complexion, and later on a tendency to hæmorrhages appears. A brown patch of pigmentation on the nasal side of the conjunctiva is not uncommon. The blood shows a leucopenia.

The disorder is now known to be due to the accumulation of lipoid in the cells of the reticulo-endothelial system, which become distended and clear. These "Gaucher cells" may accumulate into nests of a pale yellow colour, and they are found not only in the spleen but also in the liver, lymphatic glands, and bone-marrow, where they may cause atrophy of the bone and lead to spontaneous fracture.

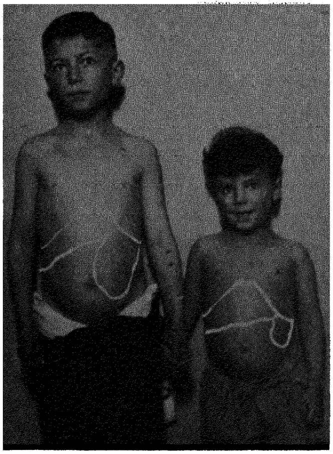

FIG. 22. Two brothers with Gaucher's disease. The outlines of the liver and spleen are indicated.

The diagnosis is not difficult if more than one member of a family is involved. The age incidence is sufficient to distinguish it from Niemann-Pick's disease. The chief difficulty is in telling it from Banti's disease, although in the latter condition the spleen does not attain to so great a size. The diagnosis can be confirmed by finding the typical Gaucher cells in the material obtained from puncture of the spleen.

The course is a long one over several years, and may go on to adult life. There is no curative treatment, but if the spleen becomes so large as to cause discomfort, or if hæmorrhages occur, splenectomy should be undertaken.

Niemann-Pick Disease

This rare condition is usually met with in Jewish children, and may affect more than one member of a family. Girls are more often affected than boys.

Symptoms appear within a few weeks or months of birth.

There is considerable enlargement of the spleen, which may reach below the umbilicus, the liver becomes enlarged, and the child becomes wasted and pale. Death occurs before the end of the second year. There is no curative treatment.

Histological examination of the liver and spleen shows the presence of large " foam cells " distended with a yellowish lipoid. Similar cells are also to be found in the bone-marrow, suprarenal cortex, and lymphatic glands, while the ganglion cells of the nervous system may also be stuffed with lipoid.

In addition to the symptoms already described, a large proportion of the cases show mental degeneration, spastic paralysis, fits, and blindness, in short the symptoms of amaurotic family idiocy (see p. 548), and further, the pathology of the nervous system in both diseases is identical. It seems probable that Niemann-Pick disease and amaurotic family idiocy are merely different clinical expressions of one and the same condition.

The Schüller-Christian Syndrome

This rare condition affects boys more often than girls, and usually appears between two and eight years of age. The youngest case occurred in an infant of eight months. The symptoms comprise a patchy necrosis of the bones, particularly those of the vault of the skull, although the pelvic and limb bones may be involved ; exophthalmos ; diabetes insipidus ; sometimes an arrest of development of the " Frohlich " type (see p. 475) ; and xanthomatosis of the skin. The necrotic areas in the skull may be masked by soft fluctuant swellings.

The disease is due to cellular accumulations of cholesterol in the affected areas. Although usually fatal, a gradual recovery has been recorded.

GOUT

This is very rare in children. The symptoms are similar to those of adults, although a swollen, red, and painful big-toe joint is not sufficient to establish the diagnosis, for acute rheumatism may pick out this joint without other joints being affected.

A case observed by the author and regarded as gout was that of a girl eleven years old, who was stated to have suffered from the age of four years from intermittent attacks of pain in the right metatarso-phalangeal joint. Each attack began as a rule at night, and for three days the joint was swollen, red, and acutely tender. No other joints were involved. The child was heavily built, the blood pressure was raised to 150 mm. Hg.

systolic, and 100 mm. Hg. diastolic, and the blood uric acid was 5·85 mgm. per 100 c.c. (normal 2–4 mgm.). A grandfather had suffered from gout for many years. The child was given a long course of vinum colchici, and while on this was free from attacks.

AMYLOID DISEASE

Amyloid disease arises as a complication of prolonged suppuration, and so may be met with in such conditions as chronic osteomyelitis, chronic empyema, or tuberculous disease of bone with sinus formation. The condition is to-day a rarity. It is a form of pathological degeneration in which amyloid material is deposited in the walls of the arterioles. The organs principally affected are the spleen, which becomes large and smooth (sago spleen), the liver, which also enlarges, and the kidneys. The urine is pale, with a low specific gravity, and contains much albumen. Anæmia, ascites, œdema of the feet, and diarrhœa may also be present.

Although the appearance of amyloid degeneration makes the prognosis of the primary illness unfavourable, a gradual recovery is possible.

LIPODYSTROPHIA PROGRESSIVA

This condition is characterised by a progressive loss of subcutaneous fat from the face and upper part of the trunk. The cause is unknown. The disease has a gradual onset in the middle years of childhood, and advice is likely to be sought on account of the cachectic appearance. Girls are more often affected than boys.

The facies is so typical that if one case has been seen, other instances can be immediately recognised. The fat gradually drains away from the face until the outline of the facial bones can be made out, the cheeks are hollow, and the appearance can only be described as gaunt. As a rule the fat of the trunk also disappears down to about the level of the umbilicus, and the

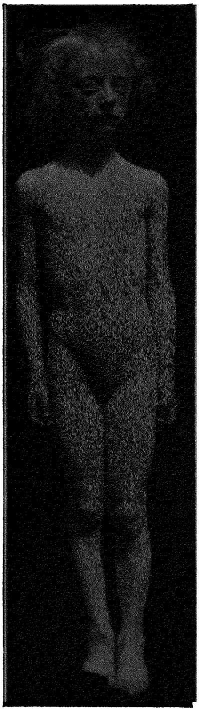

FIG. 23. Lipodystrophia in a girl aged 10 years.

arms may also be involved, but the lower part of the trunk and the legs remain unaffected.

There is no effective treatment. The disorder is likely to continue into adult life, but the general bodily health and mentality remain unimpaired.

OBESITY

A precise definition of obesity is impossible for the reason that one cannot say just where a big child ends and an obese child begins. After infancy it is more important to consider the weight in relation to the height than to the age, for obviously a tall child will only be well proportioned when the weight is several pounds above that of a shorter child of the same age. Obesity only calls for treatment when the fatness causes general comment, and gives rise to unnatural inertia or to undue dyspnœa on exertion. There is no doubt that obesity sometimes runs in families, and although this may, of course, be due to a general air of gluttony pervading the household and encouraged by the parents, this is by no means always the case. These children are inherently big, but are as a rule perfectly fit and active in spite of their weight.

There is a practical advantage in attempting to divide obese children into two groups—those whose obesity arises for exogenous reasons, and those in whom an endogenous factor plays the chief *rôle*. By far the majority of fat children belong to the former group, and efficient dieting can do much to reduce their condition, generally without the aid of drugs. Likely enough there is also an endogenous factor at work in these children, metabolic rather than endocrinal, which allows them to get through their day's activities with a minimal expenditure of energy, and so leaves available a larger proportion of their diet to be stored in the tissues. This state of affairs may be regarded as the opposite of that which obtains in highly-strung "nervous" children, who are usually thin, and whose tissues maintain such a vigorous metabolism that efforts to fatten them up are fruitless.

Exogenous Obesity. Simple overfeeding is the most important factor. This is sometimes fostered by the parents, who take comfort from their child's big appetite, allowing him to eat of whatever he likes not only at mealtimes but often between meals as well, and recounting with pride what he "gets through," although by the time the obesity has passed beyond control they may be loth to admit the dimensions of his appetite. This was illustrated by the mother of a very fat little girl, who averred that the appetite was quite slender, but when the father arrived at the end of the consultation he admitted that " she regularly eats half a loaf of bread at each meal."

Overeating brings other symptoms in its train. The increasing weight interferes with the child's activities, and so the expenditure of energy falls and the child becomes fatter, setting up a vicious circle. Further, the increasing deposition of subcutaneous fat lessens the loss of body heat, which leaves more fuel available for storage. Shortness of breath during exertion soon appears, and the child is likely to complain of aches and pains in the legs, often associated with some degree of flat-feet.

The fat is diffusely distributed over the face, trunk, and limbs, which is in contrast to the particular deposits over the pectorals, round the loins, and in the anterior abdominal wall in children with dyspituitarism (Frohlich's syndrome). Too much should not be made, however, of the clinical value attaching to the distribution of the fat, for it is only a rough guide and may be misleading.

Endogenous Obesity. At the risk of repetition it may again be pointed out that endogenous obesity is much less common than the exogenous type. The various forms of endogenous obesity are more fully considered under diseases of the endocrine glands, and include dystrophia adiposo-genitalis (Frohlich), hypothyroidism, eunuchoidism, and the obesity in association with basophil adenoma of the pituitary and hypertrophy of the suprarenal cortex. It may be noted that in the various members of this group the appetite is often noticeably small.

Treatment. The treatment of obesity in children is almost entirely a question of diet, and in this connection it may be pointed out that half-measures unwillingly undertaken are worse than useless, for not only does the child not lose weight, but dietetic restrictions are soon discarded as being of no value. If dietetic measures are to be a success the proper co-operation of the parents and the child must first of all be won, and then a dietetic policy must be steadily pursued. That being so, it must first be decided that the degree of obesity is one which really calls for reduction, and secondly the diet must be such that the child's natural growth and development is not interfered with.

The principles of the diet should be to conserve the intake of protein, and fats should be allowed for their vitamin value. Starches, on account of their water-binding properties in the tissues, should be excluded as far as possible, but an adequate amount of vitamin C must be assured by allowing green vegetables, salads and fresh fruit. These requirements will be obtained by removing from the diet : bread (replaced by crispbreads, Ryvita, McVita, etc.), potato, root vegetables (carrots, turnips, etc.), peas and beans, farinaceous puddings, flour, cocoa, chocolate, sweets. Three meals a day should suffice, although a light supper can be given to older children, and there should be no extras between meals. The following specimen diet shows how these instructions can be carried out.

BREAKFAST. No cereals. Give grape-fruit or fruit juice, followed by bacon or egg or fish. Crispbread with butter and marmalade. Milk or weak tea to drink.

DINNER. Meat soups, meat, or fish or egg. Green vegetables or salads. Followed by stewed fruit and junket. Custards and milk puddings should only be given on three days a week. Water to drink.

TEA. Crispbread with butter, honey, or cheese. Fresh fruit. Sponge-fingers. No cake or biscuits. Milk or weak tea to drink.

SUPPER (if necessary). Meat soups or bovril or fresh fruit.

The weight should be taken each fortnight, and the dietetic restrictions should only be continued for two or three months, or until the child's weight and appearance begin to conform to the normal.

A valuable addition to the above directions is to institute a fasting-day once a fortnight. It is essential that the child should spend this day in bed, and the diet should then be restricted to fluids—water, lemonade, fruit juice, or weak tea, and a little fresh fruit such as two or three oranges. The fast-day is most conveniently carried out during the week-end so as not to interfere with schooling, and too, at a time when others are at home to help entertain the child. A fast-day accelerates the drop in weight, but should not be repeated on more than three or four occasions, and is only recommended for the more gross cases.

In addition to dietetic restrictions a regular amount of exercise each day should be arranged. This must not be sufficient to make the child breathless, but should be gradually increased as the weight diminishes. Set physical exercises serve this purpose very well, and should be carried out morning and evening, and in addition the child should be encouraged to join in the school games and sports. A course of massage to the trunk and limbs is often helpful at the beginning of treatment.

Drugs play practically no part in the treatment of obesity. Small doses of thyroid are often ordered, but unless there is some clinical evidence of hypothyroidism the effect is negligible, and moreover drugs may undermine the proper carrying out of the diet. Thyroid is, of course, invaluable in reducing the obesity that accompanies hypothyroidism.

INFANTILISM

Strictly speaking, the term infantilism implies that the development of a child, along both physical and mental lines, has not kept pace with the age. Mental retardation alone does not amount to infantilism.

Infantilism is uncommon. The cases may be divided into three groups: (1) those due to endocrine insufficiency, (2) those associated with chronic disease of vital organs, (3) those for which no cause can be found.

Infantilism the result of insufficient endocrine activity is best exemplified by cretinism, in which stunting of stature and mental retardation are the result of congenital insufficiency of the thyroid. Progeria, and Simmond's disease, two conditions which arise from a deficient action of the anterior lobe of the pituitary, are further examples of this group.

Infantilism caused by chronic disease of vital organs may assume various forms. Thus renal dwarfism may complicate chronic interstitial nephritis, and the clinical picture not only includes stunting of growth but also severe genu valgum and the various signs of skeletal rickets. Hepatic infantilism occurs with congenital hepatic cirrhosis, and the children are short, plump, and tolerably alert. Infantilism is a constant feature of cœliac disease, and an instance of pancreatic infantilism has been recorded by Bramwell, in which considerable improvement followed the administration of pancreatic extract. Congenital malformations of the heart, bronchiectasis, and congenital syphilis, are further instances of chronic disease which may be accompanied by infantilism.

With the exception of cases due to cœliac disease and congenital syphilis, treatment in this group is without effect.

Infantilism for which no cause can be found is spoken of as "ateleiosis." Examples of this condition are commonly found among circus troupes. They are in fact miniature adults, for the proportions of the body are in the same ratio as in normal adults, the parts being simply of midget size. The intelligence varies considerably, and in some respects may attain to a normal adult level. In some, the appearance of the secondary sexual characters is delayed, but in others the genitalia mature in proportionate size to the rest of the body, and some of these true dwarfs are capable of procreation and may transmit the condition to their offspring.

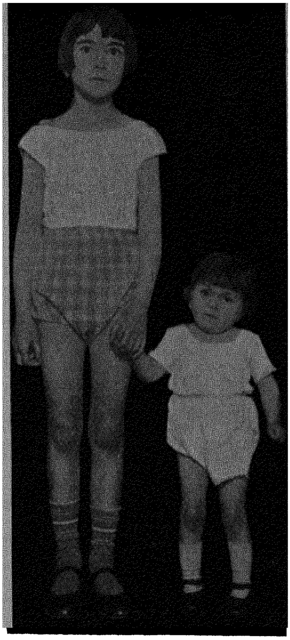

FIG. 24. Two children aged eight years. The girl on the left is of normal height while the one on the right is severely dwarfed, and weighed only 16 lbs.

CHAPTER IX

DISEASES OF THE MOUTH and ŒSOPHAGUS

STOMATITIS

This is a common affection among children of all ages, and may occur in varying degrees of severity from the simplest catarrhal inflammation to gangrene.

Catarrhal Stomatitis

This is frequently met with in conditions of under-nourishment, and often accompanies the acute specific fevers, notably measles. The buccal mucosa appears congested and is a brighter colour than usual, the tongue is furred, and there is loss of appetite. Treatment consists of frequent mouth washes with some weak antiseptic solution such as glycothymoline or a mixture of potassium chlorate gr. x., glycerin ʒi., and water to ℥i.

Aphthous Stomatitis

This is most often seen in young children under four years of age. Several small slightly raised yellowish-white patches are to be seen on the lips and buccal mucosa opposite the teeth and on the dorsum of the tongue. The patches are soon shed, leaving shallow painful ulcers. The lips are swollen and the face may appear slightly puffy, but the amount of constitutional disturbance is relatively slight. The temperature is usually raised two or three degrees, and the child is likely to be irritable, miserable, and without appetite. In many cases there is a preceding history of constipation or chronic indigestion.

Ulcerative Stomatitis

This is often a further stage of aphthous stomatitis, and is sometimes associated with dental caries or with a history of irregular action of the bowels. The ulcers are multiple and usually shallow, and occur particularly along the alveolar margins, just inside the lips, and opposite the teeth. In severe cases the gums are swollen and bleed readily, and the teeth may even be loosened in their sockets. The tongue is heavily furred, the breath is offensive, and the mouth is tender.

General symptoms consist of two or three degrees of fever, irritability, refusal of food, vomiting, and often considerable pallor. The condition is but rarely fatal, and usually responds quickly to treatment. Bacteriological examination generally shows only the normal flora of the mouth, but in some cases the mouth is infected with Vincent's fusiform bacillus and spirillum. The possibility of the stomatitis being due to the administration of mercury must be borne in mind.

Treatment. The treatment of aphthous and ulcerative stomatitis consists of frequent mouth washes with hydrogen peroxide (25 per cent.), or the inside of the mouth may be gently swabbed before and after meals with glycerin and borax. Finely powdered burnt alum may be applied to the ulcers with a camel's hair brush. When infection with Vincent's organisms is present the local application of an arsenical paint usually leads to a rapid cure ; the prescription is :

℞ Liquoris arsenicalis ʒii.
Vini ipecacuanhæ ʒii.
Glycerini ʒii.
Aquæ ʒii.

Four or five drops to be applied to the ulcers with a camel's hair brush. In addition to local treatment, potassium chlorate should be given internally every four hours, a suitable mixture being :

Potassii chloratis gr. ii.
Glycerini m. x.
Aquam ad ʒi.

So long as the mouth is tender, all food should be given cool. When the lesions have healed, the child should be sent away for a change of air for two or three weeks, and should be given a tonic such as Parrish's food.

Gangrenous Stomatitis (Noma)

This is fortunately rare, for it carries a high mortality. It is met with in severely debilitated children, and may complicate the specific fevers, notably measles. It appears on the inner side of the cheek, beginning as a large deep ulcer which slowly spreads by necrosis of the adjacent tissues until the whole cheek becomes swollen and brawny. There is severe constitutional disturbance, the temperature is raised to 104° F. or higher, and the child appears profoundly toxic. Death follows after about a week.

Careful attention to the hygiene of the mouth, particularly during the acute specific fevers, will go far towards preventing gangrenous stomatitis. Once it has developed the child should be isolated, and the affected area should be excised.

Thrush

This is due to a parasitic infection of the mucous membrane of the mouth by the fungus *Saccharmyces albicans.* The spores of the fungus are present in the atmosphere, and are conveyed to the mouth chiefly by imperfectly cleansed feeding bottles and teats. The condition is usually seen in infancy, and is much more frequent among bottle-fed infants than in those reared from the breast. Under-nourished infants are especially susceptible because of the dryness of the mouth, and for this reason also the condition often complicates infantile diarrhœa.

The infection appears as numerous small white flakes on the dorsum of the tongue, the inner side of the cheeks, and on the palate. At first sight the patches may be mistaken for flecks of milk, but they are firmly adherent to the mucosa while milk flecks can easily be rubbed away. When the infection is heavy the flakes may coalesce into a membrane, which should be easily distinguished from a diphtheritic membrane by its colour and by the fact that the general disturbance to health is much less, and also if the membrane is removed the mucosa beneath is not left ulcerated and bleeding. The general reaction is but slight. The temperature is scarcely raised and the cervical glands are not enlarged, but the mouth may be so tender that the infant cries when he is fed and may refuse to suck. If the condition is allowed to persist diarrhœa soon follows.

Treatment. Scrupulous attention should be paid to the cleanliness of the feeding bottle and teats, which must be boiled after each feed and then put to stand in boiled water until the next feed is due. If the infant is breast fed, the mother should swab her nipples with a weak boracic solution before and after each feed. The infant's mouth should be gently cleaned with borax and glycerin before and after each feed, the lotion being applied on gauze wrapped round the finger, or may be painted thrice daily with 1 per cent. aqueous solution of gentian violet.

Agranulocytic Angina : Agranulocytosis

This condition, which was first described by Schultz [1] in 1922, is characterised by necrotic ulceration of the mucosa of the mouth or fauces, accompanied by high fever and severe constitutional disturbance, and associated with a remarkable reduction in the number of polymorphonuclear leucocytes (granulocytes), which may disappear completely from the circulation. The initial infection need not necessarily be in the throat, for instances have been reported after necrotic lesions of the skin and of the respiratory and alimentary tracts. The local lymphatic glands are usually enlarged.

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[1] Schultz, W., *Deut. Med. Woch.*, 1922, 48, 1495.

Evidence is accumulating that many of the cases are caused by an idiosyncrasy to the drug Amidopyrin, which is present in many proprietary remedies. Since the Poisons Lists Confirmation Order 1936, Amidopyrin may only be dispensed on a written order.

The only instance of this condition under the author's care occurred in a girl aged eleven years, who had been under treatment for lymphadenoma for twelve months. The child was then admitted to King's College Hospital on account of high fever. The right tonsil and neighbouring structures were much swollen and ulcerated, and in a few days the whole area became gangrenous. Anæmia developed rapidly, and the total leucocytes fell to less than 100 per c.mm. Death occurred after the illness had lasted a week.

Previous to treatment with pentose nucleotide the mortality was very high, death occurring after an illness of about ten days. The dose recommended is 10 c.c. (containing 0·7 gm. of the drug) given intramuscularly twice a day until the white cell count has begun to rise, and then once a day until the count has reached normal for three days. The drug stimulates the bone marrow to produce granulocytes, but about five days must elapse after the beginning of treatment before the blood count shows any improvement.

THE TONGUE

Macroglossia

This condition is due to lymphangiomatous enlargement of the tongue. It dates from birth and may cause much difficulty in sucking ; the tongue grows rapidly and may soon be so enormous that it constantly protrudes between the gums. Treatment is surgical, and consists of the removal of the lymphangiomatous portion.

A large tongue is also one of the characteristic features of cretinism.

Tongue-Tie

This is much less common than is usually supposed. It is due to shortening of the frænum, which may also be attached nearer the tip of the tongue than is usual, causing the tip to be slightly forked. In severe cases sucking may be interfered with, and later on the speech may be affected. The effect of tongue-tie is to prevent the tongue from being protruded ; if the child is able to put out the tip of the tongue between the lips tongue-tie is either not present or is so slight as to need no treatment.

Treatment consists of dividing the frænum.

Tongue-swallowing

This is occasionally met with during the early weeks of life, particularly in feeble infants, and when the frænum of the tongue is unusually long. The tongue then falls backwards over the epiglottis so as to obstruct respiration, and this may give rise to sudden asphyxial attacks. It is not unlikely that some of the sudden deaths that occur in infancy come about in this way. Infants who have survived an attack of tongue-swallowing should be under constant observation for a few weeks until they become stronger, and should lie on their side. Fortunately as the infant grows older the tendency to tongue-swallowing disappears.

Geographical Tongue

In this condition the tongue is persistently and heavily furred, but there are patches from which the furring has disappeared, leaving the surface red and dry. The resemblance of such a patchy tongue to a map accounts for the name "geographical tongue." Although the patches may shift their position, the peculiar appearance of the tongue is likely to last for several months. Invariably there is an association with chronic indigestion, to which treatment should be directed (see p. 198). The tongue itself does not call for special treatment, but will gradually clear up as the state of the digestion improves.

THE TEETH

Teething

Although the eruption of teeth is a physiological process it is generally a cause of discomfort and often of pain, and in sensitive infants may produce a variety of symptoms. In the mildest form the symptoms consist of irritability and fretfulness, lasting perhaps for two or three days and disappearing as soon as the tooth penetrates the gum. Dribbling is a common forerunner. Some infants will give expression to their feelings by constantly rubbing the head to and fro on the pillow, others try to cram their fingers or fists into their mouth, and these symptoms are so common that parents expect them. Sometimes more alarming symptoms appear.

Fever of two or three degrees is often present and may continue for a few days, or may persist as an evening rise for some weeks, and is then likely to cause considerable anxiety lest some more serious condition than teething is present. The temperature generally falls with the eruption of a tooth, but when several teeth are cut in succession the fever may, of course, continue. In other infants the pain and tenderness of the gums leads to a loss of appetite which occasionally amounts to an absolute refusal of food, and then the weight may either remain stationary or may slowly fall. Under these circumstances the child may have to be fed entirely from a spoon. Feeding is often made easier by giving a grain of chloral before the meal.

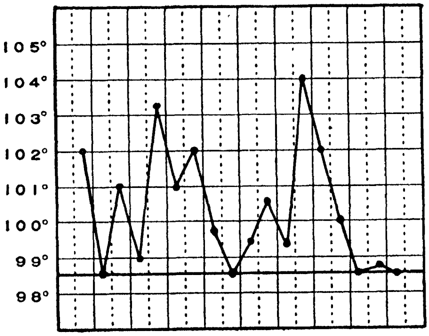

Fig 25 Temperature chart of a girl aged ten months. The temperature subsided with the eruption of two incisor teeth.

It is a common thing for parents to say that their child "cut his teeth with bronchitis" or with diarrhœa, and undoubtedly teething is often associated with an increased liability to catarrhs of the respiratory and alimentary tracts. As a rule such catarrhs are mild and respond quickly to simple measures, but the symptoms are likely to recur with each successive tooth.

Rarely symptoms serious enough to suggest some profound nervous disorder may arise. The author has seen the clinical picture of tuberculous meningitis closely mimicked by a child eighteen months old whose gums were swollen and angry-looking preparatory to the eruption of the canine and molar teeth. The child was wasting from refusal of food, and was constipated and irritable, and showed stiffness of the neck muscles, Kernig's sign, a tâche cerebrale, and photophobia. But the cerebro-spinal fluid was normal, and with the eruption of several teeth the child slowly regained his normal health. In excitable sensitive infants convulsions may sometimes precede the appearance of a tooth.

By the time the second dentition is reached, the child has become more stable, and teething disorders are much less common, but a mild persistent pyrexia may be baffling until it subsides with the eruption of a tooth.

Great care is needed before symptoms of disordered health are attributed to teething. A thorough examination must be made to exclude other conditions. Otitis media, acute pyelitis, infantile scurvy, and the convulsions of rickets are possible sources of error that must not be overlooked.

Management. When irritability and sleeplessness are the principal features, sedatives such as chloral or phenazone should be given two or three times a day until the offending tooth is erupted. Spoon feeding will sometimes succeed when a bottle is absolutely refused. When symptoms are so severe that the infant seems in danger of exhaustion or the weight is rapidly falling, relief may be given by lancing the gum just over the tooth. The value of this is illustrated in the accompanying chart of an infant whose loss of weight was attributed to teething. The gums over four teeth were much swollen and congested, and as soon as these were lanced the baby's condition improved. In practice, however, lancing of the gums is not often necessary.

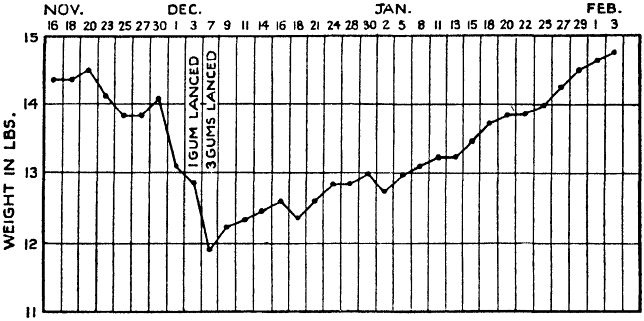

FIG. 26. Weight chart of an infant aged six months, showing the loss of weight due to teething and the improvement that followed lancing the gums over four teeth.

Teeth-grinding

This habit occurs in association with several conditions. It may be heard when there is chronic disease of the brain or meninges, particularly in tuberculous meningitis, and with hydrocephalus following upon posterior basic meningitis, and it is sometimes an accompaniment of mental deficiency. Toothache, either from caries or from the irritation of dentition, accounts for other cases, and it may also be a symptom of chronic indigestion.

It is particularly likely to occur at night, and may be so persistent as to wear down the teeth almost to the gums. It is popularly supposed to be a sign of threadworms, but this is seldom the case, and in fact when teeth-grinding and threadworm infection occur together it is probable that both are dependent on an underlying disorder of digestion.

Treatment must depend upon the cause. Conditions such as toothache and chronic indigestion can be dealt with, but nothing will check the grinding that is associated with organic disease of the brain.

Dental Caries

To anyone working much with children, the frequency of caries of the teeth must be most striking. It is often surprising how much decay of the teeth of the first dentition may exist without complaint of toothache, and this may perhaps account for the frequency with which parents are ignorant of, or disregard, the fact that their children's teeth are decayed. A routine examination of a child should always include a careful scrutiny of the teeth, for not uncommonly one or more abscesses in the gum at the roots of the teeth will be found quite unexpectedly, and not until these have been dealt with will the child's health improve. There can be no doubt that disease of the milk teeth gives rise to much disorder of health. Unexplained temperatures, loss of appetite, and symptoms of chronic indigestion may all owe their origin to dental disease, while irritation of the nervous system arising directly from dental caries may find expression in such complaints as grimacing and tic-movements of the face or such disturbances of sleep as night-terrors and enuresis. Infection of the tooth socket by streptococci and other organisms is not uncommon, and may lead not only to the formation of abscesses in the gums, but may cause such distant effects as acute hæmorrhagic nephritis and rheumatic carditis. It has been the author's experience to see both of these conditions secondary to dental abscesses.

Much importance attaches to the regular inspection of the teeth of young children, for competent treatment of early caries can do much to arrest further decay. It is surely a false doctrine to allow the teeth of the first dentition to rot away on the ground that they are after all only temporary teeth, for thorough mastication is at no time more important than in the growing child, and as decay advances so does mastication become less effective. It happens not uncommonly that caries of the first teeth is allowed to progress to such a degree that eventually the only possible dental treatment consists of multiple extractions, and the child is then left all but edentulous until such time as the permanent teeth erupt. Meanwhile his diet has perforce to consist of soft pappy food, and almost invariably his state of nutrition suffers sadly.

The work of M. Mellanby and others in the last few years has established the fact that much dental decay is undoubtedly preventable, and the application of this new knowledge should lead to a considerable lessening of dental troubles in the children of the future. The prevention of dental caries should properly begin at a time when the teeth are still forming within the jaw and are undergoing calcification, because the better the teeth at the time of eruption the more able are they to resist caries. It has been shown that diets which contain adequate supplies of vitamin D greatly stimulate the calcification of teeth, while cereal diets, particularly oatmeal, lead to the formation of hypoplastic poorly calcified teeth.

The teeth of the first dentition begin to calcify as early as the fifth month of intrauterine life, and from then on calcification progresses rapidly. It follows that in order to ensure the eruption of a well-calcified set of milk teeth attention should be directed to the maternal diet, seeing that the mother during her pregnancy does not undergo a shortage of such foods as milk, eggs, and butter, which contain vitamin D. But the prevention of caries in the first dentition does not stop there, for it has been shown that the initiation and spread of caries in children can be limited by incorporating vitamin D in their diet, while on the other hand, a cereal diet with a low vitamin content tends to have the opposite effect.

If we consider for the moment only the milk teeth, it is clear that the care of the teeth means, among other things, the supervision of the diet, not only in infancy but during the whole of the pre-school period, to see that the diet shall not be composed mainly of cereals (which on account of their cheapness and bulkiness are apt to be the principal constituent of the diet in the poorer homes), but that the amount of cereal shall be adequately balanced by foods containing vitamin D. During these years a child should have a pint of milk a day, three eggs a week, and at least half an ounce of butter a day. If the home circumstances are such that a deficient diet is likely, a teaspoonful of cod-liver oil emulsion should be given twice a day during the winter months.

The remarks concerning the prevention of caries in the first dentition apply with even greater emphasis to the permanent dentition. The first tooth of the second dentition begins to calcify a short time before birth, and except for the wisdom teeth all the remainder begin to calcify during the first two years, and calcification goes on throughout the pre-school period. If, then, during this time the diet is supervised along the lines indicated above, not only will caries of the first dentition be prevented, but the best foundation will be laid for ensuring a healthy set of permanent teeth.

It must not be supposed that by giving attention to the diet, the customary hygiene of the teeth—such as cleaning them twice a day—may be neglected. But it is not uncommon to see children whose teeth, in spite of scrupulous cleaning, are badly decayed, while others who have not taken the same care have a good set of strong teeth. No amount of cleaning can undo the harm wrought by a bad diet.

Hereditary Ectodermal Dysplasia

This rare condition is characterised by a failure of development of most, if not all, of the teeth, together with scantiness of the hair and an absence of sweat glands.

The teeth of both first and second dentitions are affected. Only the four canine teeth may erupt, or perhaps there may be one or two molar teeth as well. X-ray examination shows that there are no other teeth lying unerupted in the jaws. The jaws are correspondingly slender, the upper and lower jaw are too close together, and the cheeks are hollow, the face resembling that of edentulous old age. This appearance is enhanced by the all but bald scalp and the scanty eyebrows. The skin is also thinned and lacks pigment, while the absence of sweat glands and often rudimentary sebaceous glands makes the skin very dry. The nails may show a deforming hypertrophy.

The influence of heredity is apparent in many of the cases, for examination of the parents often shows one of them to possess similar defects to those seen in the child, although usually to a less extent.

There is no treatment.

THE ŒSOPHAGUS

Congenital Atresia

In this condition the lumen of the œsophagus is completely

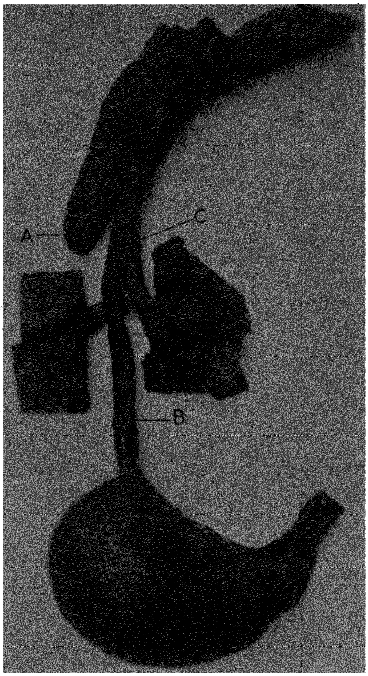

FIG. 27. Congenital atresia of the œsophagus. The upper half (A) terminates blindly, the lower portion (B) communicates with the trachea (C).

obliterated over a short section of its course, usually opposite the bifurcation of the trachea. The upper segment of the œsophagus is dilated, while the lower portion is narrow but pervious. A communication exists between the trachea and the lower portion of the œsophagus in 90 per cent. of the cases, and the presence of this communication may sometimes be suspected during life by the filling of the stomach with air during respiration.

Symptoms. These appear as soon as the infant is first put to the breast, and are so alarming and characteristic that the diagnosis can seldom be in doubt. After swallowing a few mouthfuls of milk the infant suddenly begins to cough and choke, and milk and mucus freely mixed with air-bubbles froth up out of the mouth. The infant quickly becomes cyanosed, and seems likely to suffocate, but as a rule the attack terminates by a powerful vomit, the contents of the œsophagus being ejected from the nose as well as from the mouth. The attack is repeated with each attempt at feeding. Except for the passage of a little meconium the bowels are absolutely constipated.

Attempts to feed by passing a tube into the stomach fail because the tube simply coils up in the upper half of the œsophagus. If the diagnosis is in doubt, an X-ray picture may be taken after giving a small teaspoonful of bismuth, when the level of obstruction will be revealed.

Death is inevitable, and may come about from the aspiration of milk into the lungs, causing pneumonia, or from starvation, death occurring at any time during the first fortnight. Gastrostomy, and jejunostomy, combined in some cases with crushing of the tracheo-œsophageal communication, has been attempted but has not met with success.

Congenital Narrowing (Stenosis) of the Œsophagus

This condition occurs in two forms. In the first, the lumen of the œsophagus is narrowed for a variable length, generally about half an inch or so. This may occur at any level, but is most common about halfway between the level of the bifurcation of the trachea and the œsophageal opening in the diaphragm, opposite the seventh thoracic vertebra. The investigations of Brown Kelly [1] and others have shown that the correct explanation of some of these cases lies in a failure of the œsophagus to grow to its proper length. The level of the stenosis represents the junction of the œsophagus and the stomach, the passage below the narrowing being lined with gastric mucosa, and in fact consisting of a cone of stomach drawn up through the diaphragm into the thorax—a condition termed " thoracic stomach."

In the other variety, which is very rare, a thin incomplete membrane stretches across the œsophagus, usually at some point in its lower half.

The condition is perhaps more common in boys, for of 10 cases collected by the author and Dr. Ogilvie,[2] 8 were boys. No less than five of them were prematurely born.

Symptoms. The most prominent symptom is vomiting, beginning at any time during the first year. It may date from birth, but is often delayed until semi-solids are added to the diet. The vomiting is effortless, and occurs during or soon after a meal, the vomitus being alkaline in reaction and consisting of undigested food and mucus. As the œsophagus above the narrowing becomes more and more dilated the amount of the vomit increases, and before long may obviously consist of more than the last meal. The

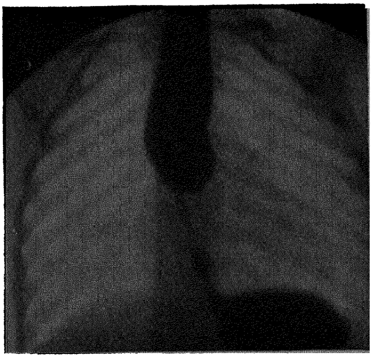

FIG. 28. X-ray of a boy four years old showing congenital stenosis of the œsophagus at the level of the seventh thoracic vertebra.

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[1] A. Brown Kelly, *Jour. Laryngology and Otology*, 1930, **45**, 680.

[2] Sheldon W., and Ogilvie, A. G., *Arch. Dis. Child.*, 1929, **4**, 347.

vomiting is subject to exacerbations lasting a week or so when both solids and fluids are returned, and then for two or three weeks it only occurs occasionally, after solid food has been taken. There may also be complaints of noisy borborygmi, and of violent eructations. It should be noted that the children experience greater difficulty in swallowing and retaining solids than liquids.

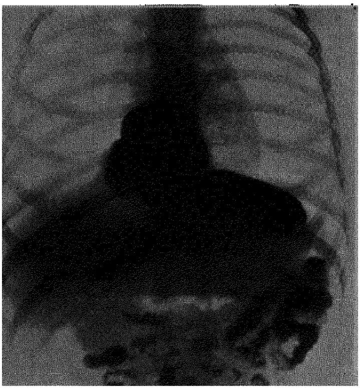

FIG. 29. X-ray of a girl four years old showing a cone of stomach drawn up into the chest—"thoracic stomach"—associated with congenital shortening of the œsophagus. The X-ray was taken with the child inverted.

The constant return of food leads to a considerable degree of starvation; the height and weight gradually drop much below the normal, and constipation is usual. Confirmation of the diagnosis may be obtained by X-raying the child after giving a solid bismuth meal such as a bismuth biscuit, which can be seen held up at the level of the obstruction.

It seems likely that a spasmodic element may play a part in some cases. This is suggested by the periodic exacerbations of the symptoms and by the fact that the vomiting will sometimes cease during any acute intercurrent illness.

Course and Prognosis. The condition generally lasts for several years. A gradual cessation of symptoms may come about in three or four years, provided that the narrowing has not been injured by bougies. Of the ten cases previously referred to, two were fatal before

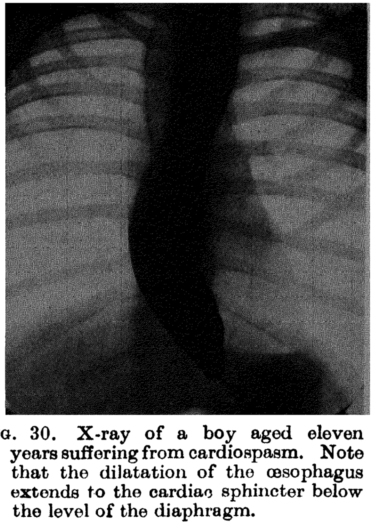

FIG. 30. X-ray of a boy aged eleven years suffering from cardiospasm. Note that the dilatation of the œsophagus extends to the cardiac sphincter below the level of the diaphragm.

five years of age, while four cases gradually lost their symptoms.

Treatment. This will depend upon the nature of the obstruction, which should be determined by a preliminary œsophagoscopic examination. If a thin diaphragm is found stretching across the œsophagus it should be ruptured. When the œsophagoscope reveals a simple narrowing the question of bougie treatment will arise. The repeated passage of bougies is very likely to injure the mucosa, leading to interstitial inflammation of the walls of the œsophagus, and eventually converting what was at first a simple narrowing into a tough fibrous stricture. It is better to allow a natural improvement to come about by simply insisting on the continued use of a fluid diet, and not to attempt any local treatment. Broths, soups, milk, and cereal foods will form the basis of the diet, together with the regular addition of vitamins in the form of fresh fruit juices and cod-liver oil. Considerable patience and perseverance is called for in the management of these cases, as several years must elapse before the child eventually recovers.

The degree to which recovery may come about may be illustrated by the case of a doctor, whose child of two years suffered from this complaint. The father, a man in the thirties, had himself suffered from the same condition all his life, and knew to a nicety how much solid food he could tolerate. He led an active life as a country practitioner. Bougie treatment had often been suggested, but had never been carried out.

Cardiospasm

The passage of food into the stomach may be held up owing to disordered neuro-muscular control of the cardiac sphincter of the œsophagus, leading either to spasmodic closure of the sphincter or, as Hurst has suggested, to a failure of the sphincter to relax when the peristaltic waves of the œsophagus reach it (achalasia). In some of the cases, psychological factors may operate initially. Thus, a girl of ten years improved considerably while under the influence of suggestion ; and anæsthesia of the palate—a common symptom of hysteria—is not infrequently present. The condition has been recorded soon after birth, but the majority of cases occur in adults, and if children are affected it is generally in the later years of childhood.

The symptoms are similar to those met with in congenital narrowing of the œsophagus, with the exception that fluid food may cause greater difficulty than solids. X-ray examination shows the level of obstruction to be at the cardiac orifice below the diaphragm, and the œsophagus may be much dilated.

Treatment. Successful results have attended the daily or weekly passage of heavy mercury-filled œsophageal bougies. It is therefore important to differentiate between congenital narrowing of the œsophagus and cardiospasm, since bougie treatment is not indicated in the former condition. The onset later in childhood, the greater difficulty with fluid food, and the level of the obstruction as shown by X-ray examination, are features that distinguish cardiospasm.

Acquired stricture of the œsophagus is said to be more common in childhood than in later life, and is generally due to swallowing some irritant or caustic poison. The stricture occurs as a rule in the upper half of the œsophagus, and treatment should be directed to dilating it by the repeated passage of thick rubber bougies.

CHAPTER X

DISEASES OF THE DIGESTIVE SYSTEM

CONGENITAL ABNORMALITIES

Congenital Intestinal Atresia

CONGENITAL atresia may occur at any level in the intestinal tract, but very rarely affects the stomach, in fact atresia at the pylorus has only been recorded on four occasions.

Duodenum

One of the most frequent situations is the duodenum ; as a rule the obstruction is in the neighbourhood of the Ampulla of Vater, but may also occur at the duodeno-jejunal junction. The symptoms arise immediately after birth, and closely resemble those of hypertrophic pyloric stenosis. There is vigorous projectile vomiting, and if the obstruction is situated below the level of the entrance of the bile duct there will be bile in the vomit. The stools are small, consisting only of meconium, and cease altogether after a few days. Progressive wasting is also a constant feature. On examination the epigastrium may be distended, and gastric peristalsis can be made out passing from left to right across the upper part of the abdomen. This may suggest a diagnosis of pyloric stenosis, but the facts that the symptoms date directly from birth, that there is no palpable tumour at the pylorus, and that the vomit may contain bile, serve as distinguishing features. If the abdomen is X-rayed after a small feed of bismuth, the level of the obstruction in the duodenum can be easily demonstrated.

At the level of the atresia the bowel may simply consist of a narrow impervious ring, or a long strip of the duodenum may be represented as a fibrous cord. Occasionally the duodenum is completely divided into two or more parts. Instances have also been recorded in which obstruction has been due to compression of the duodenum by peritoneal bands.

Treatment lies in operation. If the obstruction can be relieved the infant may recover, but the chance is a slender one, particularly if, as not infrequently is the case, there are further points of obliteration lower in the intestine.

Small and Large Intestine

Atresia may occur in the jejunum, ileum, or colon, and gives rise to symptoms of complete intestinal obstruction dating from birth. Vomiting begins during the first day or two and is associated with absolute constipation. Within a few days the abdomen becomes distended, and intestinal peristalsis can be made out. The weight steadily drops, and death occurs after two or three weeks.

Surgical treatment offers the only hope, and if there happens to be only one level of atresia a short-circuiting operation may prove successful. Unfortunately, however, the lumen of the bowel may be obliterated at more than one place, or there may be long strips of intestine consisting simply of impervious fibrous strands.

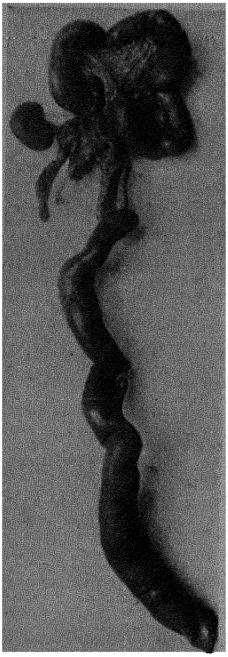

FIG. 31. Multiple atresia of the jejunum.

Imperforate Anus

The presence of absolute constipation from birth is the principal symptom. In some cases the rectum stops just short of the normal aperture and then operative treatment is relatively simple, but in others the rectum terminates some inches above the anus. A rare deformity is one in which the rectum turns forwards to open by a small orifice into the urethra, or into the vault of the vagina.

Abnormalities of the Umbilicus

The stump of the umbilical cord normally separates by a process of dry gangrene on about the fifth day, leaving a small ulcer which quickly heals. Great care needs to be taken to prevent the umbilicus from becoming infected, and it should be freely dressed with an antiseptic powder such as equal parts of boracic, zinc and starch. Occasionally a mild degree of sepsis occurs and may give rise to a soft red fleshy mass of granulation tissue. This should be treated with caustic, and a simple antiseptic powder applied.

Developmental abnormalities of the umbilicus are not uncommon. The most serious is that in which the normal return of the mid-gut within the abdomen does not take place, and the abdominal wall does not properly close (exomphalos). At birth the umbilicus is replaced by a large tumour containing intestine, which can be seen through the thin peritoneal covering. Operative treatment is usually required, but in the absence of intestinal obstruction conservative treatment by simply protecting the sac from injury has been followed by slow natural repair.

Even when the umbilicus closes normally, abnormalities may develop in connection with a persistent Meckel's diverticulum, which connects the intestine with the umbilicus, or with a persistent urachus, which connects the bladder with the umbilicus. Either of these structures may remain patent throughout their whole length, and then there will be a persistent fæcal or urinary fistula at the umbilicus, or they may remain patent only at the umbilical end, in which case a small fistula discharging a thin mucoid fluid will be present, or they may become sealed off at their ends while remaining patent in their centre, and this will give rise to the formation of a cyst either of Meckel's diverticulum or of the urachus. The treatment of these congenital deformities is surgical.

THE STOMACH

Acute Gastritis

Acute gastro-enteritis of infants has already been considered in Chapter V. In older children acute gastritis is almost always the outcome of some indiscretion of diet, of which a too free indulgence in unripe fruit or ice cream are not infrequent examples. Exceptionally it is due to the swallowing of some poisonous material in mistake for an edible substance.

Simple acute gastritis has a brisk onset, with vomiting as an early and prominent symptom. The vomiting usually lasts two or three days, the vomited material consisting at first of the indigestible food matter, and then of a good deal of mucus perhaps streaked with bright blood. In more severe cases even water may be returned, and after a day or so the child becomes drowsy, acetone bodies appear in the urine, and there may be a smell of acetone in the breath. In young children one or more convulsions may occur at the beginning of the attack.

As a rule the bowels are loose, and the stools may contain undigested food. Pain of a colicky nature is generally complained of, and is localised over the epigastrium, and there may also be some tenderness on palpation over the stomach. The tongue becomes coated with a white fur, the breath may become offensive, and the appetite is temporarily lost. The temperature is likely to be raised two or three degrees.

Treatment. When vomiting and retching are incessant, relief can most rapidly be given by washing out the stomach with a solution of sodium bicarbonate (one drachm to one pint), and this may be repeated once or twice if necessary. After the lavage a dose of castor oil should be given to rid the child as quickly as possible of whatever has brought on the attack. One teaspoonful should be allowed for each year of age. From the beginning of the attack simple fluids should be given in plenty, water, lemonade, or barley water being suitable, and these should all be liberally sweetened with glucose. Except for simple fluids, nothing in the way of food should be given until the vomiting has ceased, and then milk, milk puddings, toast and plain biscuits should be given for a day or two before the normal diet is resumed.

As to drugs, the most useful is a mixture of bismuth and soda, with the addition of belladonna if there is much colicky pain. The following mixture may be given every four hours during the first day and then thrice daily :—

℞ Bismuthi carbonatis gr. x.
Sodii bicarbonatis gr. v.
Tincturæ belladonnæ m. iii.
Pulveris cretæ aromatici gr. iiss.
Mucilaginis acaciæ m. xv.
Aquam chloroformi ad ℨii.

Peptic Ulcer

Ulcers in the stomach and duodenum are admittedly rare in children, although Theile[1] in 1919 was able to collect 248 examples. They fall into the following groups :—

(1) Ulcers in the newborn. These have been considered in connection with hæmatemesis neonatorum (p. 30).

(2) Gastric and duodenal ulcers may arise in connection with septic conditions elsewhere. They are occasionally associated with attacks of appendicitis, and have been recorded in such conditions as osteomyelitis and neonatal pemphigus. Shallow ulcers are also sometimes found at autopsy in infants who have died after long wasting illnesses associated with vomiting and

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[1] Theile, P., *Erg. d. Med. u. Kinderhk.*, Berlin, 1919, **16**, 302.

diarrhœa, but the appearance of the ulcers indicates that they are of recent formation, and not the cause of the wasting.

(3) Ulceration of the duodenum following upon superficial burns is also well known, and uræmia is another toxic condition which is occasionally accompanied by gastric or colonic ulceration.

(4) Chronic simple ulcer. This form of ulcer corresponds to the type met with in adults, and occurs more often in the stomach than in the duodenum. It is generally circular, with shelving edges, and may penetrate to any depth in the stomach wall. An instance of chronic gastric ulcer has been recorded in an infant of three months (Nixon).

Symptoms. Because of its rarity a diagnosis of ulceration is seldom made unless hæmorrhage occurs—either hæmatemesis or the passage of tarry stools. Complaints of vomiting of blood are occasionally met with in a children's Out-patient Department, but almost always a careful examination of the nose and throat shows that the blood has come from there and has been swallowed and then vomited. When, however, the occurrence of true hæmatemesis has led to a diagnosis of ulceration, a review of the history will often bring to light such suggestive symptoms as abdominal pain associated with meals and relieved by vomiting. An instance of this was a boy aged six who was admitted to hospital with a history that during the last twenty-four hours he had vomited blood several times. During the next few days several tarry motions were passed. He was treated on the usual lines for gastric ulcer and made an uneventful recovery, but the history showed that during the previous year he had suffered from bouts of epigastric pain coming on at mealtimes and lasting for an hour or so before being relieved by vomiting.

Perforation is such a rare accident that a correct diagnosis before operation is not likely to be made, although this has been accomplished in isolated instances. Cicatricial contraction of a pyloric ulcer with dilatation of the stomach is also very rare, but occurred in a girl aged seven years on whom a gastro-enterostomy was performed with complete relief of her symptoms (Miller).

Treatment. The treatment is the same as for adults. When bleeding has occurred the child should be nursed flat in bed, a small dose of opium should be administered, and ice may be given to suck. Fluids should be supplied rectally for a day or two. The diet needs to be gradually built up, peptonised milk being given to start with, followed by milk and eggs, and then slowly back to a greater variety of nourishing foods. Of drugs, a combination of alkalies with belladonna is most useful.

Gastromegaly due to Duodenal Ileus

Under this name Miller[1] has described a condition of chronic distension of the stomach caused by compression and obstruction of the third part of the duodenum where it is crossed by the root of the mesentery. The symptoms begin in infancy, when it is noticed that only small meals can be tolerated without vomiting, while in later years the stomach becomes more and more dilated and hypertrophied. Recurrent attacks of vomiting occur and are often accompanied by the passage of large amounts of mucus in the stools. Examination of the abdomen shows a fulness of the epigastrium, the area of gastric resonance is unusually extensive, splashing sounds can easily be elicited, and broad waves of peristalsis may be seen passing over the epigastrium.

As a rule treatment should be on medical lines, although in a severe case under the author's care a gastro-jejunostomy proved successful. The meals must be small and frequent, and should be given as dry as possible, drinks being taken between meals. Fermentable starchy foods and fats should be restricted, and an alkaline tonic, such as a mixture of rhubarb, nux vomica, and soda, may be given before meals.

THE INTESTINE

Chronic Indigestion

Chronic indigestion in one form or another is one of the most common ailments of childhood. It not infrequently makes its first appearance soon after the period of weaning from a purely milk diet, at a time when the variety of the diet is enlarging and fresh foods are being tried, and when it is easy to overtax a child's digestion, especially by giving an excess of bulky foods such as cereals, raw fruits, and vegetables. The time of the second dentition, roughly between the sixth and tenth years, is another age-period in which chronic indigestion is commonly met with. It may also be pointed out that if a child is already being fed to the limit of his digestive capacity, any debilitating illness may be enough to cause a breakdown of digestion, and in this way be the beginning of prolonged ill health. Many years ago Eustace Smith pointed out the particularly bad influence of whooping cough in this respect, and measles is almost as harmful.

Causes. *Unsuitable Food.* Generally the error lies in giving too much fermentable starchy food—potato, bread, and pastry are common offenders in this respect. The surfeit of starch undergoes bacterial fermentation in the small bowel, and leads to gaseous distension and colic. The contents of the small bowel are prematurely urged into the colon, and thence evacuated as offensive undigested motions.

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[1] Miller, R., *Arch. Dis. Child.*, 1930, **5**, 83.

Another source of trouble are the foods which leave a good deal of residue after digestion. These foods are generally rich in cellulose, and include the root vegetables, such as carrots and turnips ; peas, beans, and raw fruit, particularly when the skin is eaten ; nuts, coarse oatmeal, and wholemeal breads. The residue from this type of diet is bulky and irritating to the colon, which reacts by secreting large amounts of mucus. Peristaltic movements then become less effective, constipation may follow, and the stools are large and contain masses of " white jelly " or mucus.

Mismanagement. Many of the troubles of chronic intestinal indigestion can be traced to incorrect training and mismanagement in the home, and a careful inquiry into the child's daily routine should always be made. Parents often worry far too much about their child's appetite—particularly in the case of an only child—and resort to coaxing and bribery to make him eat up his food. Children are not slow to take advantage of this, finding that it well repays them to be difficult and finicky at mealtimes. Irregular feeding between mealtimes is often the result, the appetite becomes capricious, and a taste for indigestible articles of food is likely to develop. Mismanagement sometimes appears for the first time when schooling begins. The rush in the morning to get the child off to school is likely to mean a too hurried breakfast, with scarcely time for evacuation of the bowels. Other children whose school is too far away for them to return home at midday have to make a lunch of sandwiches, which is never satisfactory, and then are likely to be given a heavy meal at home in the evening, which almost always leads to digestive disturbances. Eating between meals, particularly of sweets and biscuits, impairs the appetite and constitutes another important factor. Then, too, there is no doubt that in some children the stress of working for school examinations, scholarships, and prizes, militates against good digestion.

Chronic Naso-pharyngeal Infection. Chronic naso-pharyngeal infection is a frequently associated condition. Chronically infected tonsils and adenoids, or a constant muco-purulent catarrh of the pharynx, are likely to lead directly to a chronic gastro-intestinal catarrh with the symptoms of a disordered digestion. A thorough examination of the throat in a good light should always be made, since treatment confined to the digestive tract is not likely to be successful if an unhealthy state of the throat is allowed to persist.

Early and extensive caries of the first dentition is at times a factor of considerable importance. Unless young children are able to masticate their food properly, they bolt it in lumps, and this leads to colic and a poor digestion. Periodic examination of the teeth of young children is of great importance, provided that it is coupled with treatment carried out as soon as caries begins to appear. One sometimes sees children in whom caries has been allowed to progress to such a pitch that a wholesale extraction of most of the first dentition has been deemed necessary. The benefit to health by losing so many decayed teeth is often more than counterbalanced by the interference with digestion which loss of the masticatory apparatus entails. Many of these unfortunate edentulous children, unable to take solid food, have to be fed on broths and soft cereal foods, which makes it impossible for them to grow adequately or to find sufficient energy to carry them through an ordinary day's programme.

Symptoms. While in many cases the entire alimentary tract is affected, in others the brunt of the disturbance falls on some particular part, and the symptoms vary accordingly. Occasionally the symptoms are mainly gastric (atonic gastritis) ; the child frequently eructates, and often vomits a great deal of mucus, and there may be epigastric pain, or at least a feeling of fulness and discomfort. At other times the small intestine is chiefly at fault and the stools are offensive and loose ; or the colon may be principally affected, and then the motions are likely to contain an excessive amount of mucus (catarrhal or mucous colitis).

The onset of chronic indigestion is gradual, and the child is likely to be brought for advice because he is not " getting on " or because he is irritable, easily exhausted, has turns in which he goes " deathly white," is puffy under the eyes, or sleeps badly.

The appetite is as a rule bad, and the child " doesn't eat a thing." Occasionally the opposite is the case and the appetite is enormous, but the food is so badly digested and poorly absorbed that it fails to make the child thrive. Often there is a craving for just those articles which are likely to aggravate the condition, such as sweets, raw fruits and raw vegetables ; severe perversion of the appetite, in which such odd things as coal, paper, earth and so on are relished—the condition of dirt-eating or *pica*—is at times a feature.

The bowels may be either constipated or loose. The stools are brown or greenish, offensive, often large and at times loose, and may contain lumps of undigested food. An excess of mucus in the stool has already been commented upon. Threadworm infection is a common coincidental trouble, because the unhealthy state of the bowel makes such a suitable nidus for the development of the worms. Abdominal pains are frequent, but seldom severe ; they are generally localised round the umbilicus and tend to be worse towards the end of the day. Vomiting of food is not a prominent symptom, except in the "atonic gastritis" type, but the child often retches in the early morning and brings up the mucus which has collected in the stomach overnight.

Apart from the gastro-intestinal tract there may be symptoms referable to almost every other system. Instability of vascular tone is indicated by sudden attacks of pallor, and sometimes fainting. The extremities are cold and often of a raw red colour, or have a bluish tinge. In many cases the temperature rises one or two degrees in the evening, or may remain slightly raised throughout the day, and this may continue for weeks or months. This low fever often passes unnoticed, which is perhaps as well, because once it is discovered it is likely to cause constant anxiety to the parents, who find it difficult to believe that a chronic digestive disorder is sufficient reason for the temperature remaining up for such a long time.

Vague pains in the limbs are a common complaint. They tend to occur towards the end of the day, and are related to fatigue, disappearing when the child rests ; their distribution is in the legs rather than the arms, and they are not so obviously related to atmospheric changes as are rheumatic pains. A morning cough is not unusual, and is often relieved after the child has brought up mucus from the stomach.

Nervous symptoms are very common. Rapid fatigue, irritability, and lassitude are usual. Headache is often complained of, and may be present at any time of day. The most troublesome nervous manifestations, however, are those which affect sleep. Often the child lies awake for hours, and when sleep eventually comes it is fitful, and broken by tossing and twitching. Sleep walking and night terrors are likely, and attacks of croup may occur during the night—laryngitis stridulosa. Nocturnal enuresis is also a common trouble.

Physical examination shows the child to be below average weight. Loss of postural tone may be evident by the child's weary stance, with his sagging abdomen, exaggerated lumbar lordosis, prominent scapulæ, drooping shoulders and flat chest. There may be orthostatic albuminuria. The complexion is sallow, there may be dark rings round the eyes, and the lower eyelids may be so puffy as at first to suggest nephritis. Occasionally the backs of the hands and feet may be puffy. The skin may be either dry or greasy, and in long-standing cases the hair becomes thin, dry and brittle. Skin lesions of an urticarial nature are often present.

The tongue is as a rule covered with a thin brown fur, and at times the furring is patchy, a red glazed surface showing between the patches of fur. Because of its resemblance to a map, this type of tongue is often called "geographical." The teeth are often carious and may be considerably ground down, particularly the incisors. The abdomen is soft, flabby and distended, but not tender, and the liver can generally be felt about two fingersbreadth below the costal margin. When the stomach is principally affected there is much distension of the epigastrium, the area of gastric resonance is enlarged, and splashing sounds may be obtained over the stomach. The urine is often described as high-coloured, or may be cloudy with phosphates.

Prognosis. Death from chronic indigestion is quite exceptional, but when it occurs, post-mortem examination reveals no anatomical lesion. The entire absence of any morbid anatomy is, however, what one would expect in a disease the nature of which is a disorganisation of function, not of structure. The prognosis is eventually good, but almost always several months of diligent treatment are needed before any real improvement is brought about.

Treatment. The two essentials in treatment are the general management of the child and the selection of a proper diet. Drugs are helpful, but are only of secondary importance.

(1) *General Management.* As a rule rest in bed is not necessary, but in the more severe cases with much abdominal distension and loss of weight preliminary treatment in bed for three or four weeks may be advisable. Otherwise the child should get up after breakfast and should be out in the open air as much as possible.

Meals must be taken at regular times and should be not at all hurried, and feeding between meals must be absolutely vetoed. As a rule it is better for the meals to be taken dry, drinks being given between meals, and this is especially so in the type with much gastric distension. Late meals just before bed-time must be particularly avoided.

During the day a regular amount of exercise is necessary, but it should not be too violent nor tiring, and it is better for the child to be out playing under the supervision of an adult. A rest at midday is important, but it must be a proper rest, either on a couch or actually lying down on the bed. This should begin half an hour before the midday meal and should continue for an hour afterwards. Again at tea-time the child should rest for a quarter of an hour before tea and for half an hour after. Obviously under these circumstances the child cannot attend school, at any rate not during the first month or so of treatment. Bed-time should be early—a child of six or seven years of age should be in bed by half-past six.

Foods not to be taken	Foods that may be taken
Coarse oatmeal.	Fish (boiled).
Wholemeal and brown bread.	Chicken. Rabbit.
White bread should be toasted.	Meat should be minced.
Raw fruit.	Meat soups and broths.
Root vegetables (carrots, turnips, parsnips, swedes).	Crisp toast and rusks.
Peas and beans.	Crisply fried bacon. Otherwise no fried food.
Sweets and chocolates.	Milk, eggs, butter. Milk puddings.
Jam with seeds or pips.	Honey, seedless jam and jellies.
	Fruit stewed, and fruit juices.
	Green vegetables puréed (cauliflower, cabbage, spinach).
	Potato (see note below).

Note: Potato should be well boiled, not mashed, and limited to two tablespoonfuls.

Stewed fruit: a little daily, provided that no skins or pips are given.

Each morning before breakfast the child should have a warm bath followed by a cool sponge, which, as the child gets used to it, should be changed to a cold douche, and this should be followed by a vigorous rub down with a rough towel. A course of general massage is beneficial. Particular attention must be paid to the teeth, caries being dealt with by fillings rather than extraction whenever the condition allows. When the tonsils are diseased they should be removed.

(2) *Diet.* The points to receive attention in the diet are that starchy foods should be reduced, and foods that are likely to leave a bulky residue after digestion should be avoided. The above table indicates the lines along which the diet should be managed.

(3) *Drugs*. Speaking generally, the most useful drugs are mixtures of alkalies with a bitter tonic, and they should be taken before each meal. A useful prescription is :

℞. Sodii bicarbonatis gr. v.
Pulveris rhei gr. ss.
Tincturæ nucis vomicæ m. ii.
Spiritus chloroformi m. ii.
Infusi gentianæ compositi ad ʒii.

The alkali promotes secretion of gastric juice and may help to loosen any mucus from the gastric mucosa ; the rhubarb has a mild laxative action and at the same time improves the tone of the bowel wall ; the nux vomica is given for its general tonic effect, while the gentian stimulates the appetite. Mercury is also sometimes useful, and is best given as calomel, using small doses such as one-sixth of a grain thrice daily. When there is much atony of the stomach, and the appetite is poor, the following acid mixture may with advantage be given in sips with meals :

℞. Acidi hydrochlorici diluti m. v.
Acidi phosphorici diluti m. ii.
Glycerini m. x.
Aquam ad ʒii.

to be taken in water during meals.

After treatment. Generally an improvement begins to appear after three or four weeks of the above *régime*. At this stage a period of convalescence at the seaside for six or eight weeks is often invaluable ; there is no better way of accelerating the recovery. While the child is away the routine of daily management and the dietetic precautions should not be relaxed ; and generally, if drugs are being used, they should continue to be taken. Also at this stage, if anæmia has been a troublesome symptom, small doses of iron should be given ; a useful form is the syrup of the iodide of iron, of which half a teaspoonful may be taken with the same amount of malt extract after meals. Iron should not be given during the earlier stage, as it is apt to be badly tolerated when the digestion is out of order. No attempt should be made to feed up the child, and the parents must be particularly warned against this. Cod-liver oil, or one of the numerous preparations containing it, is a favourite prescription of the parents who are anxious to get their child on, but it often affects the appetite adversely.

The various associated complaints, such as night terrors, nocturnal enuresis, or threadworm infection, may also call for treatment, and a course of breathing exercises, or exercises to improve the tone of the postural muscles, is often beneficial.

Cœliac Disease

This condition was first described by Gee in 1888 in a paper entitled " On the Cœliac Affection," and although the titles " Acholia " (Cheadle) and " Intestinal Infantilism " (Herter) have since been employed, they have been given up in favour of the name " Cœliac Disease." It is a chronic disorder which begins in early childhood, and is characterised by diarrhœa, the stools being large, fatty, and offensive, and by severe wasting and eventually much interference with growth. Though not a common condition, it is met with in all grades of society, and affects girls a little more often than boys.

Symptoms. The onset is a gradual one between about nine months and two years of age, at a time when children are being weaned from a milk diet on to a variety of foods, and therefore at a time when mild digestive upsets are common. Because of this, cœliac disease is at its onset likely to be mistaken for some simple digestive disturbance, and it is only when the usual remedies fail and the child slowly drifts towards emaciation that the real nature of the condition comes to be realised. In the early stages loss of appetite is prominent, only a few ounces of food being taken at meals and that very unwillingly, the motions become loose, attacks of vomiting occur, and the child becomes moody and irritable, continually grizzling and resenting examination, although no actual pain or tenderness can be discovered. The temperature may be subnormal, but is as often raised a degree or two for months at a time, and at intervals may rise three or four degrees.

When the disease is fully developed the most noticeable feature is the profound emaciation. This is so extreme that the child has the appearance of being merely skin and bone, and compared with the pinched face, thin chest, and stick-like limbs the distended and tympanitic abdomen affords a striking contrast. The abdominal wall is, however, as devoid of fat as other parts, and is so tightly stretched over the distended intestines that peristaltic movements can often be seen through it. The loss of fat is also very obvious in the buttocks, where the skin hangs in loose folds. The complexion becomes sallow, the forehead appears wrinkled, and the hair becomes thinned and dry and loses its lustre. Curiously enough, the tongue is not as a rule furred—in contrast to chronic intestinal indigestion—but the breath may be sour.

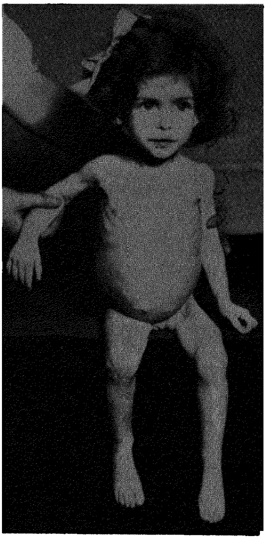

FIG. 32. Cœliac disease in a girl aged two and a half years. Her weight was only 14 lbs.

The abdomen is soft and can be easily palpated, which helps to distinguish it from the doughy abdomen of tuberculous peritonitis, but otherwise the only unusual feature is the smallness of the liver, which may be completely hidden behind the ribs. This has a clinical bearing, for it offers one point in differentiating cœliac disease from chronic intestinal indigestion, in which the liver can generally be felt with ease. The distension of the abdomen is accounted for in part by the excessive fermentation going on in the intestine and in part by the tonelessness of the muscles of the abdominal wall. The loss of tone also affects the muscles of the trunk and limbs to such an extent that the child is often unable to sit up or support himself, and the ligaments become so lax that the joints can be moved beyond their normal range.

Anæmia is present in practically all cases, and may reach a severe degree. As a rule it is hypochromic in type with a low colour index, due to iron deficiency, but a megalocytic hyperchromic type of anæmia has been described in cases that have continued into adult life.

The stools of an untreated case are characteristic. They are large, unformed, of a porridgy consistence and colour, and are extremely offensive. They vary in number, seldom exceeding five or six a day, although sharp bursts of diarrhœa may occur when their number rises, while during periods of improvement they may fall to one a day. Chemical analysis of the dried stool[1] in an untreated case shows that about two-thirds of it consists of fat (normally in a young child not more than one-third is fat), although the proportion of split to unsplit fat is unaltered. Such analyses indicate that there is no failure in the mechanism for digesting fat, the trouble lies rather in a failure to absorb the fat after it has been digested.

Complications. *Œdema* is a not infrequent complication, and generally appears on the top of the feet or on the back of the hands.

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[1] Harrison, G. A., and Sheldon, W., *Arch. Dis. Child.*, 1927, **2**, 338.

Occasionally it is more widespread, and free fluid may collect in the peritoneal cavity. The œdema develops quickly, and after lasting a variable time may suddenly disappear, but beyond emphasising the severity of the condition it makes little difference to the prognosis. It has been attributed to lack of vitamin B in the diet, but the œdema may persist after any deficiency of this vitamin has been rectified. It is more probably due to a sustained high intake of salt consequent on a high protein diet, for many of the protein foods, especially fish and meat, have a high salt content. *Purpura* is less common than œdema, but is usually of more serious omen. The purpuric spots generally appear at first over the lower abdomen, in the groins, round the base of the neck, and at the edge of the axillæ, places where the skin is thrown into folds, and bruising may also develop over pressure points, and may then lead to bedsores. *Scurvy* is a third complication, which is not surprising when one considers for what long periods these children survive on an almost entirely protein diet. Unfortunately fruit juice and other dietetic sources of vitamin C are often so badly tolerated that the prevention of scurvy is a matter of considerable difficulty. *Tetany* is another complication which may be met with during the later stages of the disease, and is due to the low level of the blood calcium, which in turn is accounted for by the enforced absence of fat from the diet.

Rickets as a complication of cœliac disease has only been recognised within recent years, indeed Still has pointed out that rickets is strikingly uncommon at the beginning and during the active phase of cœliac disease. This has its explanation in the fact that growth must be taking place for rickets to occur, and during the long-drawn-out active period of cœliac disease growth ceases. It is not until recovery begins to take place and the child begins to grow that rickets is likely to appear. It only occurs in the severe cases, and then not before the age of seven, which would be about the age when severe cases, if they are to recover at all, should begin to do so. The development of rickets is due primarily to the absence of fat in the diet leading to a deficient supply of vitamin D, calcium, and phosphorus—the three materials which we know to be necessary for the prevention of infantile rickets. Of the rachitic manifestations genu valgum is usual, and there is likely to be enlargement of the epiphyses and beading of the ribs, and fractures and bending of the long bones. The X-ray appearance shows a characteristic broadening and scooping out of the bone ends, with an irregular fluffy epiphyseal line, and the shafts of the bones are considerably rarefied.

Pathology. It has already been pointed out that analyses of the stools indicate a failure to absorb from the intestine the products of fat digestion. The combination of a flat blood sugar curve after giving glucose by mouth, with a high curve after intravenous glucose, shows that there is also a failure to absorb glucose from the bowel. The essential feature of cœliac disease is then a selective breakdown in the mechanism of absorption from the alimentary tract, but the reason for this is obscure. Post-mortem examination shows as a rule nothing beyond profound wasting. Various explanations have been put forward, of which none is entirely satisfactory. Obstruction to the lacteals has been suggested on an analogy with tuberculosis of the mesenteric glands, which sometimes interferes with the absorption of fat and gives rise to stools resembling those of cœliac disease, but in cœliac cases no anatomical evidence of such obstruction has been forthcoming. An insufficient secretion of bile has also been suggested, but the colour of the stools is due to excess of fatty acids and not to diminution of bile pigment, and although administration of bile salts sometimes gives a temporary improvement it does not lead to cure. Chronic intestinal infection has been suggested as a cause, and in six out of eleven cases reported by Still there was evidence of infection with dysentery bacilli, but as a rule the intestinal flora is normal. There is no evidence of a defective pancreatic function, although the clinical picture produced by cystic fibrosis of the pancreas (see p. 264) bears certain resemblances to cœliac disease.

Looking back over the symptoms and complications of this disease, it will be realised that many of them, such as anæmia, œdema, scurvy, tetany, and rickets, are the result either of impaired absorption or of deficiencies due to the restricted diet.

Diagnosis. Although in the early stage a diagnosis can scarcely be made with certainty, when the clinical picture becomes more fully established the diagnosis should not present difficulty. The condition which is most likely to be mistaken for cœliac disease is severe chronic indigestion, because it may also lead to considerable wasting and abdominal distension, but the stools are not fatty, although they often contain much mucus—which is not a feature of cœliac disease. Other points of distinction are that the liver in chronic indigestion is easily palpable, and the child makes a much better and quicker response to careful treatment. Stools similar to those of cœliac disease occasionally occur in tuberculosis of the mesenteric glands, but the wasting is less severe, stunting of growth is not so apparent, and the enlarged glands can be felt.

Course and Prognosis. Cœliac disease invariably runs a long course, the mildest cases lasting for a year or two, and others often dragging on for three to five years. The course is marked by phases of improvement which last for perhaps three or four weeks, in which the stools become less in number, are less offensive and may even tend to be formed, and the weight may rise by two or three pounds. These periods of improvement are, however, deceptive, and are very likely to encourage a too quick increase in the diet, with the unhappy result that the stools become worse again and within a week or so the weight drops back to its original level. At other times there seems no accounting for the rapid relapses. Although the weight fluctuates with the phases of improvement, there is often no appreciable gain for as long as a year at a time. The above chart of a severe case shows the weight taken on the first of each month over a period of two years, and it can be seen that at the end of this time the weight was much the same as at the beginning. This child eventually recovered.

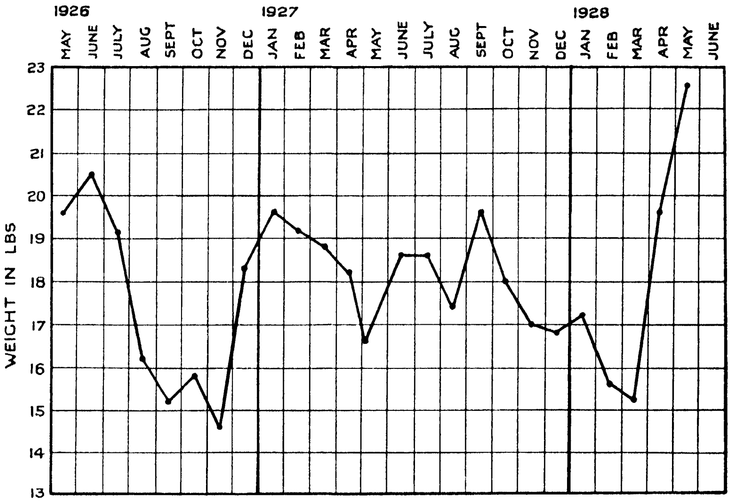

FIG 33. Weight chart of a boy with cœliac disease showing the characteristic fluctuations over a period of two years.

During the period of stationary weight, growth in height is also at a standstill, and gradually leads to a very obvious stunting.

Such a prolonged illness also leads to a lack of mental interests, and the child may come to seem considerably behind-hand for his age, but actually the mentality is unaffected, and when recovery comes about the child is able to learn quite normally.

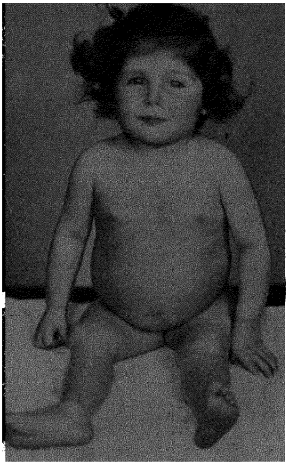

FIG. 34. Cœliac disease after recovery. This is the same child as in Fig. 32.

The mortality is about 15 per cent. Death may be due to a sudden relapse of diarrhœa or to progressive exhaustion, and any intercurrent infection such as measles or pneumonia is also badly borne by such enfeebled children and may prove fatal.

When recovery takes place, it does so gradually. Some children eventually return practically to normal, although they may remain intolerant of fat in their diet for many years. Occasionally a peculiar phase occurs in which recovery is at first marked by an excessive deposit of subcutaneous fat, the appearance of obesity being accentuated by the stunted height, but this is a temporary phase and eventually the contour becomes normal. When recovery is delayed until the disease has been present for several years, some degree of dwarfing may persist into adult life. Instances of cœliac disease in adult life have recently been described[1] in which in addition to a fatty diarrhœa there was also dwarfing, bony deformity, and severe anæmia. Such cases are rare, and the prolonged history is not improbably due to lack of treatment during the early years.

Treatment. The most important part of the treatment lies in the diet. Fat is so badly tolerated that it has to be almost entirely excluded. This particularly applies to the fat of cow's milk, so that in whatever form cow's milk is given it must be completely skimmed of its cream. Breast milk is usually tolerated quite well, but the difficulty of obtaining a prolonged supply is almost insuperable. Asses' milk has a low fat content and could be used were it not for its prohibitive cost. Unfortunately, the dietetic difficulties do not stop at the fat, for cœliac children also have much difficulty in dealing with starchy foods on account of their tendency to produce intestinal fermentation and distension with diarrhœa. These difficulties are increased if there is also an intolerance towards fresh fruit juices, for the danger of scurvy then becomes a real one. Another difficulty which often has to be contended with is the child's individual capriciousness, for although a carefully planned diet may be prescribed it is not improbable that the child will refuse to have anything to do with it.

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[1] Bennett, I., Hunter, D., Vaughan, J., *Quart. Jour. Med.*, 1932 (New Series), 603.

The diet should be built up on the following lines. Fresh milk must be as completely skimmed as possible, but it is usually better to give a dried skimmed milk, such as skimmed Cow and Gate or Horlick's Malted Milk. Egg yolk should be avoided. Protein foods form a large part of the diet, and may be given in the form of meat broths, liver broth and liver extracts, calves'-foot jelly, pounded chicken, rabbit, scraped steak, egg white, steamed fish, and such powdered proteins as Casec, Casumen, Sanatogen or Plasmon. A teaspoonful of Marmite should be given daily. Meat, fish, and Marmite contain a high salt content, and they may have to be temporarily reduced should œdema appear.

The various forms of carbohydrate need to be carefully selected, as they so readily provoke diarrhœa. Rusks, thin crisp toast, grape nuts or Force, honey, seedless jam, fruit jellies made with skimmed milk, sponge cake, and sieved green vegetables are generally among the more easily tolerated carbohydrates. Banana has quite a reputation, but the fruit should be very ripe (turning brown) and mashed through a sieve. Two or three can be given during the day if they do not upset the digestion, but they are by no means always well borne. Cereals such as cornflour, ground rice, groats, custard, etc., must be added with caution, because of their tendency to increase the diarrhœa.

From the beginning of treatment a daily ration of fresh fruit juice should be given as an anti-scorbutic, but it may have to be temporarily omitted if it provokes fresh diarrhœa. Fortunately, the recent introduction of pure vitamin C in the form of ascorbic acid has made it possible to do without fresh fruit juice. A 25-mgm. tablet of ascorbic acid should be given daily.

Although its mode of action is as yet uncertain, the benefit of crude liver extract given by injection seems undoubted. Two c.c. of Campolon should be given intramuscularly twice a week, and this may be continued over several weeks.

In the event of a severe relapse of diarrhœa, subcutaneous or intravenous salines may be required to combat the dehydration. Under these circumstances, intravenous glucose, by circumventing the failure to absorb alimentary glucose, is of particular benefit.

The general management of the child also calls for much care. Skilled nursing is essential, for a good nurse can do much to overcome the child's capriciousness in the matter of appetite. In a condition in which wasting is so prominent it is most important to preserve carefully the body heat. Woollen garments should be worn with sleeves and leggings long enough to cover the hands and feet. Fresh air is also invaluable, and in warm weather the child should be placed out of doors in a corner protected from draughts. Gentle massage is a useful means of keeping up the muscle tone and maintaining a good circulation.

Drugs are only indicated for the relief of specific symptoms. Small doses of castor oil such as three to five drops three times a day are helpful in checking diarrhœa, and may be combined with intestinal antiseptics such as salol in the following prescription :—

℞. Salolis gr. i.
Olei ricini m. iii.
Mucilaginis acaciæ m. xv.
Aquam anethi ad ℥i.

Small doses of silver nitrate such as gr. $\frac{1}{6}$ twice a day have been found useful (Hutchison). Bile salts sometimes have the effect of checking diarrhœa, $\frac{1}{2}$ gr. of sodium glycocholate and sodium taurocholate being given in a small capsule at mealtimes. A tablet of takadiastase, at mealtimes, is sometimes effective in lessening the amount of fermentation. When the appetite is so poor as to interfere with treatment an alkaline bitter mixture before meals is useful, and for this purpose the infusion of gentian with soda, or tincture of rhubarb and soda, can be used. Iron and ammonium citrate (gr. 5 ter die) should be given to combat the anæmia.

The possible development of rickets at a time when recovery is just beginning should be prevented by giving exposures to ultra-violet rays, and by adding five drops of irradiated ergosterol to the diet twice daily.

Constipation after Infancy

The regular evacuation of the bowels is as important in childhood as in adults, and depends very largely on good habits being learned during infancy. Chronic constipation in older children is most often due to a failure to acquire regular habits, and not uncommonly dates from persistent constipation in infancy. The frequency with which constipation is met with in mentally defective children is commented on elsewhere ; in them the cause also lies largely in the difficulty in teaching good habits. Left to his own devices, the average child will certainly not concern himself overmuch with the state of his bowels, naturally preferring to hasten from meals to play with his fellows or to get to his toys. When schooling begins, punctuality in the meal-times at home is essential, in order that sufficient time may be allowed for the child to empty the bowels before going to school. Much of the debility that is met with in children during their period of school life is connected with faults of this kind. Another factor sometimes at work in school children is lack of exercise, especially in those who are studying for scholarships and examinations.

Much is made of the importance of diet in the prevention and treatment of constipation, but dietetic causes are less apparent in older children than in infancy. Certain generalisations can, however, be made. Thus in an otherwise healthy child a diet which leaves too little residue will naturally tend to make the stools small. This state of affairs is often seen in older children who are receiving an unduly large amount of milk in their diet. In general terms, an insufficient intake of food leads the same way. On the other hand, the mistake is often made of varying the diet in the opposite direction by giving an excess of those foods which leave a large residue after digestion, such as coarse oatmeal, raw fruits, nuts, and root vegetables. A diet of this sort often causes, but seldom overcomes, constipation, for it more easily excites the colon to secrete large amounts of mucus, and this tends to render peristalsis ineffective. Constipation produced in this way is very likely to be accompanied by a good deal of abdominal pain from colicky contractions of the intestine.

The large bowel in early childhood is relatively longer than at other ages, and instances are occasionally met with in which the colon loops upon itself, and by its twists and kinks causes constipation. An example of this was a girl of six years who had always suffered from troublesome constipation. The abdomen was much distended with large veins coursing over its surface, and the colon was dilated and thickened and packed with fæcal masses. A barium enema showed the descending colon to be looped upon itself, accounting for the severe constipation. Occasionally constipation dates from some local lesion at the anus, such as a fissure, which has caused so much pain that the child is frightened to open the bowels. Anal stenosis and anal spasm have already been mentioned in dealing with constipation during infancy; if such conditions remain untreated constipation will persist into later childhood.

The effects of constipation are numerous. The appetite is impaired, sometimes to such an extent that it helps to keep up the condition. The skin is unhealthy and greasy, and the tongue is covered with a brownish fur. The circulation is often sluggish, and the hands are blue and cold. Nutrition is interfered with and the weight may fall considerably below the standard for the age. Not uncommonly constipation gives rise to a mild but persistent fever of a degree or so, and when the parents discover that the temperature is constantly raised they not unnaturally become anxious and magnify insignificant symptoms until fears of tuberculosis or some other such condition are entertained. It has to be remembered that a child may suffer from chronic constipation although the bowels are open once a day, for the passage of the intestinal contents may be none the less delayed in spite of the daily action.

The nervous system of a child acts in close sympathy with the health of the digestive tract, and symptoms of nervous disorder are common in children who are persistently constipated. Peevishness and complaints of headache may at times be so prominent as to lead to a mistaken suspicion of tuberculous meningitis. In infancy the occurrence of convulsions due to constipation has already been mentioned, and occasionally fits due to the same cause may occur in older children who are of a highly strung nature. Disturbances of sleep are also common enough as a result of constipation, the story being that the child tosses restlessly, is wakeful, or suffers from night terrors. Bedwetting may also be an associated symptom.

During examination the colon can generally be easily felt, often throughout its whole length, the cæcum filling the right iliac fossa with a soft gurgling tumour. When constipation is prolonged the accumulation of fæces may reach striking proportions, several hard craggy masses as big as chickens' eggs may be felt in the abdomen, particularly near the umbilicus, and may simulate very closely enlarged mesenteric glands or may even be mistaken for renal or retroperitoneal tumours. The diagnosis between fæcal masses and enlarged glands will often remain in doubt until a course of colon wash-outs and laxatives has gradually brought the masses away. It is in such severe cases as these that the complexion may become dusky or cyanosed, especially over the cheeks—enterogenous cyanosis. The colour slowly turns to a healthy rosy hue as the health of the bowel improves.

Treatment. *General Management.* Nothing is more important than the correct training of the child. A definite time each day should be set aside for the purpose of defæcation. This should be after breakfast, and if necessary after tea as well. In this connection small points, such as seeing that the child's feet reach to the floor, are important, and a footstool should be put in the lavatory so that the feet may be firmly planted. An adequate amount of exercise in the open air every day should be obtained, and if the circulation is sluggish and the skin unhealthy the daily warm bath should be followed by a cold douche and a brisk towelling. Massage and exercises to strengthen the abdominal muscles are useful when there is much distension, but for the more usual type of case the equivalent of massage should be obtained by ordinary daily exercise.

Dietetic treatment by itself is seldom sufficient to correct habitual constipation. The addition of much "roughage" such as nuts, husks, bran and raw fruit in the hope of stimulating the bowel to contract and expel its contents is attractive in theory, but is frequently disastrous in its results, for if it fails to achieve its object it leads to still further distension of the colon, which is rendered atonic, while the irritation of the mucosa simply leads to a mucous catarrh of the large bowel. In a different category are those articles of food which contain a laxative principle, such as the pulp of prunes or figs, and they may be usefully added to the diet, although reliance should not be placed solely upon them. Apart from these simple additions, the diet needs to be easily digestible and without an excess of those foods which will leave a bulky residue. It is also important to ensure a sufficient fluid intake, and it is often a good plan to give the child a glass of warm water on rising and after the midday meal.

Drugs. Drugs play a most important part in the treatment of chronic constipation in children, but it is a mistake to wait until the child is constipated and then to give a brisk purge ; much better results are to be obtained from the daily administration of drugs in laxative rather than purgative doses, and in the writer's opinion the use of drugs in this way is of greater value than dietetic measures alone. Castor oil has simply a purgative action, which is followed by constipation, and therefore should never be employed in treating chronic stasis of the bowel.

The laxative drugs may be considered under three headings.

In the first place there are the salines such as magnesium and sodium sulphate, which act by increasing the fluid bulk of the stool. Half to one teaspoonful should be taken in the early morning before breakfast. Milk of magnesia is another preparation of this kind, but its effect is more valuable in infants than in older children. Except in the mildest cases, however, saline laxatives are unsatisfactory, as the large bowel may soon become tolerant of them.

The vegetable laxatives such as senna, cascara, and aloes are often most useful because their stimulant action is delayed until the colon is reached, the passage of food to the large intestine is therefore unhurried and there is plenty of time for digestion and absorption to take place. Which particular drug should be taken is largely a matter of personal taste. An infusion of from three to eight senna pods taken at bed-time has the advantage that the action is a gentle one, is easily controlled, and the dose can be readily regulated. Cascara is most pleasantly prescribed in the form of a half teaspoonful of " Cascara Evacuant " at bed-time, and is a reliable mixture. Aloes has the disadvantage of sometimes causing rectal irritation, and the child may therefore make several visits to the lavatory. Syrup of figs is a very popular remedy, although it is usually given in purgative doses once a week, which is the wrong way to use it. Like the other drugs of this class, it should be given daily, but only in laxative doses. An equally useful method is to give a mixed prescription of small doses of these drugs after meals, combined with nux vomica for its tonic effect on the bowel wall, and with an anti-spasmodic such as hyoscyamus to prevent any colic. The following prescription in use at The Hospital for Sick Children is often successful, and should be continued for two or three months.

℞. Tincturæ nucis vomicæ m. i.
Tincturæ zingiberis m. ii.
Extracti hyoscyami gr. $\frac{1}{8}$.
Aloes gr. $\frac{1}{10}$.
Syrupi sennæ m. xv.
Aquam anethi ad ℥i.
One teaspoonful after meals.

Of the chemical laxatives mercury in small doses is the most useful. It is best given as calomel gr. $\frac{1}{8}$ three times a day, or to young children one to two grs. of grey powder may be prescribed at night. A teaspoonful of confection of sulphur at bed-time is particularly of service when the stools are hard and dry, as it quickly softens them. It is in this type of case also that paraffin finds its greatest value, but the proper dose varies with the individual. The best way to order paraffin is to start with one teaspoonful two or three times a day, and slowly increase the dose until the desired effect is obtained. Occasionally paraffin leaks away apart from the stool and soils the garments, an indication that too much is being given. The addition of gr. ¼ of phenolphthalein to a teaspoonful of paraffin acts as a laxative and is generally more useful than paraffin by itself. It is as well to explain at the outset of treatment that the use of drugs will need to be continued over two or three months in order to promote a proper habit of the bowels.

Lastly a word must be said about the use of enemata and suppositories in the treatment of chronic constipation. They are much employed. When there are large accumulations of hard fæces it is often necessary to begin treatment by giving an ounce or two of warm olive oil per rectum in order to soften the mass, following this in an hour or so by a colon lavage with warm water or warm saline, and this may have to be repeated daily for a few days. Enemata should, however, only be used to clear away large masses, and having achieved this object they should then be discontinued. The giving of an enema every day is thoroughly bad—to many children an enema is quite an abhorrent thing, while their continued use tends to encourage rather than cure a lazy habit of the bowel, and eventually the rectum becomes ballooned and the anal sphincter patulous. The occasional use of suppositories in infancy has already been considered, but in older children they should be entirely avoided.

Hirschsprung's Disease (Idiopathic Dilatation of the Colon). This remarkable condition is characterised by profound constipation and enormous distension of the abdomen, due to gross dilatation and hypertrophy of the colon. The condition was first described by Hirschsprung in 1886. There is a remarkable preponderance of cases in the male sex; of thirty instances from the records of The Hospital for Sick Children, twenty-seven were boys (Barrington-Ward.)

The most prominent symptom is constipation. This begins quite early in life and may actually date from birth. It is often so severe that the bowels may be only opened once a week, and at times even three or four weeks may pass without an action. Eventually the contents of the bowel putrefy, and for a few days large, loose, offensive motions are passed until the colon is empty,

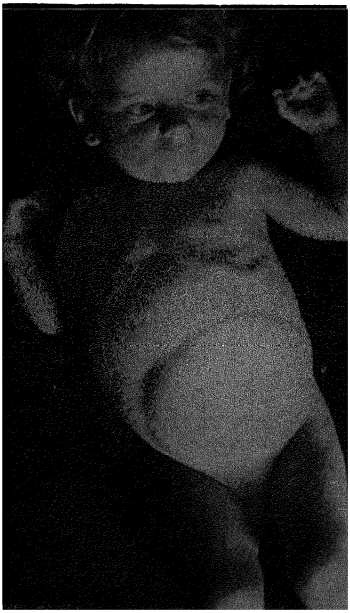

FIG. 35. Hirschsprung's Disease in a boy aged seventeen months, showing the distended colon undergoing peristalsis.

so that the history is likely to show long periods of constipation broken by brief spells of offensive diarrhœa. Considerable toxic absorption from the bowel takes place, growth is interfered with, the weight is much below the average, and the complexion is pale, or there may be some degree of enterogenous cyanosis. Loss of appetite and offensiveness of the breath are common, and occasionally vomiting occurs. Attacks of abdominal pain may be complained of, but are not usually severe.

Examination shows the abdomen to be remarkably distended. The outline of two or three loops of the greatly enlarged colon can usually be made out through the thin abdominal wall, and can be seen undergoing slow peristaltic movements. Pressure on the inferior vena cava may cause the veins of the anterior abdominal wall to dilate, and may give rise to œdema of the legs. Rectal examination may show some ballooning of the lower bowel, but fails to reveal any obstruction. The great size of the colon and the absence of any obstruction can also be demonstrated by an X-ray taken after a barium enema.

Post-mortem examination shows that the dilatation begins at the cæcum and becomes progressively greater until the pelvirectal junction is reached, where it usually stops abruptly, although occasionally the dilatation continues down to the anus. The wall of the colon is also much thickened owing to hypertrophy of its muscle. A remarkable feature is the absence of any demonstrable obstruction at the point where the dilatation ends. The mucosa is likely to be ulcerated, and there may be areas of small-celled infiltration in the sub-mucosa.

Various theories have been advanced to account for the condition. A primary muscular hyperplasia has been supposed;

and an abnormal development of neuromuscular control associated with overaction of the sympathetic innervation of the large bowel has been put forward, in support of which Cameron has demonstrated a degeneration of Auerbach's plexus at the level of the pelvi-rectal sphincter.

Prognosis. Probably more than half the cases perish from chronic toxæmia and exhaustion within the first two or three years. In others a balance seems to be reached, and with care life may be prolonged for some years, and occasionally adult life is attained.

Treatment. Medical treatment consists of emptying the bowel by repeated enemata, and is at best merely a means of reducing the fæcal accumulations. Purgative drugs seem to have very little effect, and the colon soon becomes tolerant of them. In conjunction with other treatment, the following mixture has occasionally seemed useful :—

℞. Tincturæ podophylli m. v.
Tincturæ belladonnæ m. 5.
Tincturæ nucis vomicæ m. iiss.
Aquam chloroformi ad ℥ ii.

Benefit has also been recorded from injections of pituitrin, and abdominal massage sometimes leads to temporary improvement. The diet should be as nutritious as possible, with omission of any foods that are likely to leave a bulky residue.

The older methods of surgical treatment such as colotomy and colectomy were seldom successful, and have now been abandoned. A considerable trial has been made of the relatively simpler operation of sympathectomy, the inferior mesenteric ganglion being resected. The results have varied, but in successful cases the operation has been followed by a daily action of the bowels, although diminution of the abdominal distension has not always taken place. In others, medical treatment has had to be continued after the operation, but with a greater measure of success.

The most recent treatment has been to give a spinal anæsthetic.[1] It is necessary to obtain complete anæsthesia as high as the fifth or sixth thoracic root. How this treatment exerts its effect is not properly understood, but when the anæsthetic wears off, the sympathetic and parasympathetic work in much better harmony, and a daily action of the bowels can be easily obtained. The treatment, in skilled hands, is simple, and if after some months the improvement is no longer maintained, the anæsthetic can be repeated.

[1] Telford, E. D., and Simmons, H. T., *Brit. Med. Jour.*, 1939, ii., 1224.

Acute Ulcerative Colitis: Dysentery

Acute ulcerative colitis may be met with at any age in childhood. It is at times the result of some gross dietetic insult, and also may be the outcome of prolonged constipation, but the majority of cases result from infection. Culture of the stools may show only the bacillus coli, or streptococci or organisms of the dysenteric group may be isolated. The presence of the latter cannot be judged merely by the severity of the symptoms, since they are sometimes present when the symptoms have never been anything but mild. In this country bacillary dysentery is almost invariably due either to Flexner or Sonne infection. Flexner dysentery is the more severe, but Sonne infection is the more common and is particularly troublesome in a children's ward owing to the ease with which it spreads from child to child. Flexner organisms may survive in the intestine for long periods, but Sonne bacilli seldom persist for more than two or three weeks. Agglutination tests on the blood afford a means of detecting a previous attack of dysentery.

As a rule acute colitis has a rapid onset, the temperature rises three or four degrees, and the child complains of abdominal pain. The stools quickly become loose, and may be increased in number up to a dozen or more a day. At first they contain fæcal matter, but later on consist chiefly of mucus streaked with blood and pus. Vomiting is likely to occur at the onset, but does not persist. In mild cases the symptoms settle down in three or four days, and the general health is but little disturbed, but in more severe cases dehydration and prostration appear quickly, and there may be delirium. In these cases the abdomen becomes tumid and there may be tenderness over the colon. The temperature is also likely to be higher, reaching 103° or 104° F.

At autopsy the colon appears congested, and the mucosa may be pitted with many small shallow ulcers, and may be smeared with blood and muco-pus. The mesenteric glands are acutely swollen.

Prognosis. Acute colitis may be a most serious disease during the early years, and under the name of ileocolitis has already been mentioned when dealing with diarrhœa in infancy (p. 97). In older children recovery generally comes about in from three days up to two or three weeks, and not uncommonly the diarrhœa is followed by a short period of constipation. The course is, however, often marked by one or more relapses, which are generally less severe than the original illness.

Treatment. The child must be confined to bed, and if the organisms of the dysentery group are recovered from the stools the child should be isolated and treated on infectious precautions. During the acute stage the diet must be a fluid one, consisting principally of water, barley water, or lemonade, together with milk, arrowroot, meat broths and jellies. If dehydration is severe it should be dealt with by subcutaneous or intravenous salines.

In the early stages the colon may be gently irrigated with warm saline, and if the diarrhœa has been severe a small starch and opium enema is often of service. For a child of two to five years two ounces of starch enema with three to five drops of tinct. opii may be given. When bleeding is persistent, two or three ounces of a 0·5 per cent. solution of protargol may be given as an enema. Of drugs by the mouth, opium in small doses is most valuable, but the dose needs to be regulated with care. At one year of age half a minim of tinct. opii may safely be given, and the dose may be increased by a quarter of a minim for each year.

Kaolin in teaspoonful doses thrice daily is sometimes helpful in checking the diarrhœa, or the following mixture may be employed in children over two years of age :—

℞. Bismuthi salicylatis gr. v.
Tincturæ catechu m. x.
Pulveris cretæ aromatici gr. v.
Mucilaginis acaciæ m. xx.
Aquam chloroformi ʒii.

The apple treatment of acute colitis and dysentery has proved very effective. Raw sweet apples are used ; they should be peeled and grated, the pulp and juice being fed as a mash. Each feed should be prepared separately, but whether the pulp turns brown is immaterial. Feeds of apple should be given every four hours, and for a child of one year as many as eight to twelve apples may be used in the day. The apple diet should be maintained for two or three days, by which time the stools should have become formed, and reduced in number to one or two in the day. During the next three days the diet should return to normal. So long as apple is being given, other food including milk should be entirely withheld, but fluid will be required, and may be given as water, glucose water, or preferably weak tea, allowing two and half ounces for each pound of body weight per day. If tea is given, Indian tea should be used, and should be prepared by infusing half a teaspoonful of tea in half a pint of water for two minutes only. It should be given without milk.

Good results have followed treatment with sulphanilylguanidine, a sulphonamide compound which is only slowly and incompletely absorbed, and therefore exerts its main effect on the flora of the intestine. The dose should be the same as that of other members of this group of drugs (see table on p. 715).

Anti-dysenteric serum may be given in Flexner infection, but its use in Sonne dysentery is disappointing.

Chronic Ulcerative Colitis

This is fortunately a rare condition in childhood. Most of the cases are due to a streptococcal infection of the bowel, but occasionally dysentery bacilli can be isolated from the stools. The symptoms begin as an acute colitis, but the passage of loose, offensive motions containing muco-pus and often blood persists, and there are frequent attacks of abdominal pain. The temperature remains raised, and the child becomes slowly more anæmic and wasted, until, after the illness has dragged on for several months, death may come about from sheer exhaustion. An X-ray after a barium enema shows a complete lack of haustration, the colon having a strikingly tubular appearance.

Treatment. Absolute rest in bed is essential. The diet should be as nourishing as the appetite will allow, and will consist chiefly of milk, eggs, milky foods and broths. A course of raw apple diet is often beneficial. Small doses of opium by mouth may help to check the diarrhœa, or a starch and opium enema may be given with the same object. The bowel may also be gently washed out with a hypertonic saline solution or a 1 in 5,000 solution of flavine. When bleeding is persistent, lavage with silver nitrate, 1 in 20,000, may be used. Transfusions of whole blood are often valuable as a means of conserving the child's strength and overcoming the anæmia, and they can be repeated several times, according to the child's condition. Intestinal antiseptics, such as Salol and β-Naphthol, are ineffective, but sulphanilylguanidine (see p. 715) should be given a thorough trial, as it sometimes exerts a strikingly beneficial influence. Anti-dysenteric serum should be used when dysenteric organisms are present in the stools.

Lienteric Diarrhœa

This is a condition in which attacks of colicky pain and an urgent desire to defæcate occur during meals or immediately afterwards, and may make the child rise hurriedly from the table. It is not uncommon at about the time of the second dentition. It may be found in company with the symptoms of chronic indigestion, and the stools may contain undigested food matter and mucus, but it more commonly occurs in children of a nervous constitution, and in them is the outcome of an unduly sensitive gastro-colic reflex. As a rule the general health is unimpaired.

When the condition is accompanied by symptoms of indigestion, the latter will call for treatment, but for the diarrhœa itself there is only one drug which can be relied upon, and that is opium. A convenient preparation is pulv. ipecac. co., of which 2 grs. may be given twice a day for a few days to a child of five years and upwards.

Appendicitis

Appendicitis is the most common of the acute abdominal emergencies of childhood. It may occur at any age, but is rare in infancy and is seldom met with under three years.

Symptoms. The onset is brisk. Pain is generally the first symptom and is one to which the greatest importance must be attached when taking the history. The pain is at first colicky in character, and is likely to be localised near the umbilicus. Vomiting may follow, and the lower bowel may be rapidly emptied by two or three loose motions. Within a few hours the temperature rises three or four degrees, the pulse runs up to perhaps 130 or so a minute, and the child looks pale and ill. Food is refused, and the bowels, after an initial emptying, become constipated. Vomiting, except initially, is not a prominent feature. If the child is old enough to localise his pain, the site may now shift towards the right iliac fossa.

Inspection of the abdomen shows some limitation of the normal respiratory movement, and the right iliac fossa may seem slightly full. On palpation rigidity of the anterior abdominal wall can usually be made out, most marked over the lower half of the right rectus muscle, and there may be an area of maximum tenderness about half-way between the umbilicus and right anterior superior iliac spine. When an abscess forms round the appendix, a tender mass may be felt occupying the right iliac fossa. A rectal examination should always be carried out. It may reveal tenderness localised over the right side, or may enable an abscess to be felt bimanually.

The symptoms and signs may vary from the above description according to the position of the appendix. When the inflamed organ hangs down into the pelvis it may come in contact with the bladder or rectum. Both diarrhœa and frequency of micturition are then likely, and the point of maximum tenderness is just above the pubis. Or the appendix may be in a retro-colic position behind the ascending colon, and the maximum tenderness will then be in the right loin just above the iliac crest. There may also be spasm of the ilio-psoas muscle causing the right hip to be persistently flexed.

The most important complication of acute appendicitis is perforation, leading to general peritonitis. The injudicious use of purgatives during the early stages, or even a too vigorous palpation over the appendicular area, materially increases the risk of perforation. Should the appendix be cut off by adhesions, so that perforation is followed by a local abscess, unwise treatments such as those just indicated may rupture the abscess and spread infection over the peritoneum. The onset of peritonitis is indicated by a worsening of the child's condition; the face assumes a drawn expression, the pulse rises while the temperature may at first tend to drop, vomiting reappears, and the whole abdomen becomes very rigid and board-like. It should be remembered that perforation of the appendix may be followed by a sudden and sinister disappearance of pain.

Diagnosis. Several conditions may simulate appendicitis. A severe bilious attack or an attack of cyclical vomiting may do so, but if the initial symptoms are taken in their right order it will usually be found that vomiting and retching have preceded by some hours the appearance of pain, while in appendicitis pain is generally the first symptom. A history of similar attacks having taken place previously with perhaps a regular periodicity would indicate cyclical vomiting, but the presence of ketone bodies in the urine is of little significance, for in any feverish condition associated with vomiting or with a period of brief starvation ketone bodies may quickly appear in the urine.

Pelvic appendicitis associated with diarrhœa may need to be distinguished from pneumococcal peritonitis. Both the tenderness and the rigidity in pneumococcal cases are diffuse rather than localised to the right side, and this may be confirmed during a rectal examination. An inflamed appendix situated behind the colon may simulate acute pyelitis on account of the tenderness in the loin, but a microscopical examination of the urine for pus cells will distinguish these conditions. There may be greater difficulty in distinguishing a right-sided perinephric abscess, especially during the early stages before it has caused any bulging of the loin, for both may cause a spasmodic flexion of the hip joint, but with a perinephric abscess there is no noticeable constipation, and the tenderness is likely to be a little higher at about the junction of the quadratus lumborum and the twelfth rib. Another common source of difficulty is an early pneumonia or pleurisy on the right side, for the pain in these cases may be referred to the appendicular area, and the right side of the abdomen may be held rigid. The relative disproportion between the respiratory and pulse rates in pneumonia, and the negative findings on rectal examination, are valuable differentiating features. Even the most careful examination of the chest may at first show nothing abnormal, but generally within a few hours the air entry at the base of the right lung becomes definitely diminished. If doubt persists, it is better to wait twelve hours or so, by which time a fresh scrutiny of the case is likely to show small changes by which the correct diagnosis may be reached. Lastly it should be borne in mind that fever, vomiting, constipation and slight tenderness in the appendix area may occur with acute follicular tonsillitis, and it is probable that in these cases there is actually some swelling of the appendix. If a proper inspection of the throat is carried out in all cases of suspected appendicitis the physician is not likely to be misled.

Prognosis. The prognosis depends on the speed with which the diagnosis can be reached and proper treatment instituted. If the appendix can be removed before it has infected the general peritoneum the mortality is negligible. The outlook is not so good in very young children, partly because the condition is so rare at that age that it may be at first overlooked, and partly because the lack of the child's co-operation makes it more difficult to detect with certainty the physical signs.

The treatment is surgical.

Recurrent Appendicitis

It may be doubted whether a condition of chronic appendicitis exists, at all events in several hundred post-mortem examinations on children the writer has never seen an appendix to which this title could be given. Recurrent attacks of appendicitis do, however, occur, and may eventually culminate in an attack so severe as to call for prompt surgical relief. At first these attacks may be so mild that medical advice is not sought, the condition being regarded as due to indigestion, while in others vomiting occurs for a day or so and is associated with pain and tenderness in the right iliac fossa, but the symptoms subside with rest in bed and the application of warmth locally. Between attacks the health may seem satisfactory, but more often the child is constipated, pale. and liable to vague abdominal discomforts. Help in diagnosis may sometimes be had from a bismuth meal or a barium enema, which may show some abnormality in the filling and emptying of the appendix.

In the face of a history of recurrent attacks of this sort, with symptoms definitely referable to the right iliac fossa, it is generally wise to advise an exploratory laparotomy and to have the appendix removed. Tuberculous infection of the mesenteric glands may also give rise to pain in this region, but it should be possible to feel the enlarged glands on careful palpation.

Acute Intussusception

Acute intussusception is most common between about six months and two years of age. It is interesting that almost always the infant has previously been perfectly well, and if anything errs on the plump side.

Symptoms. The onset is sudden, and pain is the most prominent feature. The pain occurs in bouts every few minutes, and is severe enough to make the child scream out and double himself up. The importance of pains with these characters in a child who has been previously well cannot be too strongly emphasised, for they are occasionally the only symptom. The time between the attacks may at first be as long as half an hour, but the interval gradually becomes more and more shortened as the attacks succeed each other. Soon after the initial attack of pain the child usually passes a loose motion containing some mucus and bright blood, and this may be repeated two or three times. After the lower bowel has been emptied the child is usually constipated, although small motions of blood and mucus may continue to be passed. Vomiting may occur at the onset, but is by no means a regular feature. The temperature varies ; it may be normal or slightly raised, but is sometimes sub-normal.

Examination shows the infant lying quietly, with the colour drained from the face. During the bouts of pain the knees are drawn up and the child cries for a few minutes until the spasm is over. If the abdomen is palpated between attacks a tumour, which is the intussusception, can be felt somewhere along the line of the colon. As a rule it is in the region of the hepatic flexure or transverse colon, but may be anywhere between the ascending colon and the rectum. The tumour is somewhat sausage-shaped, and if palpated for some minutes it may be felt to vary in firmness, although this is a less pronounced feature in acute intussusception than in the chronic variety. At first the abdomen is only held rigid during the attacks of pain, but if treatment is delayed for two or three days the rigidity increases, vomiting may become persistent, and together with absolute constipation indicates that general peritonitis has developed.

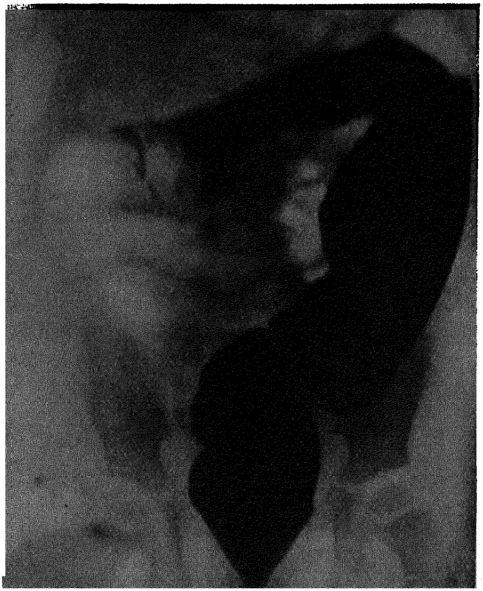

FIG. 36. Barium enema in a boy aged two and a half years, showing an acute intussusception.

A rectal examination should always be carried out. When the intussusception has extended down to the rectum its tip can be felt with the end of the finger as a mass filling the lumen of the bowel, and with a sulcus between the intussuscepted portion of intestine and the wall of the rectum. As a rule the tip of the finger stall will be found on withdrawal to be smeared with blood. It occasionally happens, however, that an intussusception is unaccompanied by bleeding, and this is particularly likely in the type which begins higher up in the small intestine. The tumour can, however, be felt in the abdomen in these cases. X-ray examination is seldom necessary in making the diagnosis, but if there is any doubt a barium enema may be given. The barium stops suddenly when it reaches the intussusception, but thin streaks of it track along between the two layers of bowel to give a typical picture (see illustration).

Diagnosis. The diagnosis is made on the history of recurrent sharp abdominal pain, the passage of blood and mucus per rectum, the palpation of a tumour in the abdomen, and the results of rectal examination. Difficulty may sometimes be experienced in distinguishing between acute intussusception and Henoch's purpura, in which sudden severe abdominal pain may be followed by the passage of blood per rectum. In the latter condition, however, there is no tumour to be felt, and further examination may show an early purpuric rash on the limbs or buttocks or perhaps in the mouth. Localised patches of œdema on the face, trunk, or limbs is also sometimes an early indication of Henoch's purpura. In acute colitis bright blood may be passed per rectum, but the onset is not so sudden, diarrhœa persists, and there is no tumour in the abdomen. The red protruding mass of a rectal prolapse differs from an intussusception in that the prolapsed mucosa is continuous with the mucosa of the anal canal, nor is the child so obviously and seriously ill.

Prognosis. The outlook depends on the rapidity with which a correct diagnosis is reached. If operation is performed within a few hours of the onset the outlook should be quite favourable, but if it is delayed for two or three days it may be found impossible at operation to reduce the intussusception, or the bowel may already have become gangrenous, making it necessary to resect the diseased portion. The outlook then becomes extremely grave.

Intussusception in some children shows an unfortunate tendency to recur, and may do so several times. In these cases there is usually some abnormality of the attachments of the mesentery allowing an undue mobility of the bowel.

Treatment. The treatment is surgical, and operation should be carried out as soon as possible after the diagnosis has been made.

Chronic Intussusception

This is much less common than the acute variety ; and is usually met with in older children. The onset begins in the same way as an acute intussusception, with recurrent attacks of abdominal pain and perhaps vomiting, but the passage of blood is less constant, which may account for the condition being at first overlooked. The circulation of the affected portion of bowel is also less disturbed, so that the intussusceptum manages to retain its blood supply, and the lumen of the bowel remains sufficiently patent to allow food to pass, so that a small motion may be voided each day. A chronic intussusception may remain for several weeks, during which time bouts of a milder pain continue, and the weight slowly falls. A history of this sort should prompt a careful examination of the abdomen for two characteristic signs. In the first place there will be a sausage-shaped tumour in the line of the colon, and patient examination enables one to feel it undergoing alternate hardening and relaxing—a feature which is almost conclusive evidence of chronic intussusception. In the second place short peristaltic waves may be observed sweeping along the colon up to the tumour and at that point fading away.

Diagnosis. The diagnosis is made on a history of recurrent pain over several weeks, beginning much as does an acute intussusception, and accompanied by progressive wasting, together with the presence of an abdominal tumour which varies in consistency and over which peristaltic waves can be made out. The diagnosis may be confirmed by X-ray examination after a barium enema, which shows a picture similar to that seen in acute intussusception. The characteristic hardening and softening of the tumour distinguishes it from other masses such as a tuberculous omentum, or fæces.

Treatment. The treatment is surgical.

Volvulus

This is an infrequent cause of intestinal obstruction in children, and is caused by a twist or rotation of the intestine either in the longitudinal axis of the bowel or more commonly round the axis of the mesentery. In some cases the bowel becomes twisted around a peritoneal adhesion, or may become rotated round a persistent Meckel's diverticulum, while other cases are associated with abnormal attachments of the mesentery. Volvulus generally affects the small intestine, and as the vascular supply is quickly cut off gangrene of the affected portion of bowel soon follows.

Symptoms. The symptoms are those of acute intestinal obstruction. The child is seized with acute abdominal pain, making him cry out and double himself up with colic. Vomiting is an early and persistent symptom, especially when the obstruction occurs high up in the small intestine. To begin with there may be one or two loose motions, but afterwards the bowels become completely constipated. On examination the abdomen is held rigidly, and is tender over the region of the volvulus. The temperature is likely to be raised, and ketone bodies soon appear in the urine. Unless the condition is quickly relieved, general peritonitis follows.

Diagnosis. Volvulus is seldom diagnosed before operation, the diagnosis not getting further than acute obstruction. Occasionally a volvulus may spontaneously untwist itself, and then may recur more than once, and in such cases there is a serious risk of mistaking the attacks for cyclic vomiting.

Prognosis. Although volvulus may resolve spontaneously, this is a rare ending and cannot be expected. The outlook depends largely on the speed with which surgical relief is undertaken. The mortality in many statistics is as high as 80 per cent. (Fraser).

Treatment. The treatment is surgical, immediate operation offering the only chance of recovery.

LOCAL LESIONS OF RECTUM AND ANUS

Rectal Prolapse

Prolapse of the rectum is not infrequently met with during the first year or two of life. It occasionally occurs as a result of tenesmus during a sharp attack of diarrhœa, or may be caused by the efforts of the bowel to expel a polyp attached to the rectal mucosa, but in the great majority there is a history of constipation, and the prolapse is due to the straining during defæcation. Such conditions as threadworms, loss of ischio-rectal fat, and phimosis, which have been advanced as possible causes, probably have little real bearing.

As a rule the history is that after the child has defæcated the rectal mucosa appears through the anus as a congested red protrusion which bleeds easily when touched, in fact the stools are likely to be tinged with blood. The prolapse can generally be replaced easily, but may recur again and again. Digital examination almost always shows the anal sphincter to be toneless. The diagnosis is, as a rule, simple, and is made from the history and the local appearance. Occasionally an intussusception may protrude from the anus, but the child is then obviously ill, the history is a short one, and on rectal examination there is a deep sulcus between the rectum and the intussusception.

Treatment. The protruding bowel should be gently sponged with cold water, and then can be replaced without difficulty. To prevent a recurrence, a soft cotton wool pad may be placed over the anus and be kept in position by a bandage or by strapping the buttocks together. If the child will co-operate, defæcation should be performed while he lies on his side. As a rule constipation requires treatment. Foods which are likely to leave an indigestible residue should be omitted from the diet, and the motions should be softened by the daily administration of paraffin, the dose being pushed until the motions are passed without straining. Small doses of strychnine should also be given to increase the tone of the bowel muscle, five drops of Easton's syrup given in water three times a day being very useful for this purpose. Even the most obstinately recurring cases can be overcome provided the treatment is carried on for a month or two. More heroic methods such as the injection of alcohol or other sclerosing fluids into the anal tissues are both painful and unnecessary.

Rectal Polyp

Rectal polyp is not an uncommon condition, but is easily overlooked. Generally the polyp is about as large as a pea or bean and is attached to the posterior wall of the rectum by a short pedicle. The polyp may be either myxomatous or adenomatous, the latter variety being sometimes hereditary and often multiple, and then rarely there may be hundreds of small polypi throughout the colon—a condition of polyposis.

A rectal polyp gives evidence of its presence by the frequent passage of small amounts of bright blood in the stools, but otherwise the child's health is scarcely affected. To make the diagnosis a digital examination of the rectum is necessary, when the polyp can be felt as a small soft lump which can be moved from side to side but cannot be pulled down, and so can be distinguished from fæces. If doubt exists, a sigmoidoscopic examination can be made.

Treatment is surgical and consists of removal of the tumour.

Piles

Piles are uncommon in childhood, and are almost always associated with chronic constipation. They are sometimes noticed soon after birth. As a rule there is a solitary external pile scarcely larger than a split pea, which may be symptomless or may be the cause of screaming when the bowels are opened, and the stools may then be streaked with blood. Treatment must include the relief of constipation, while as a local application some such astringent ointment as ung. hamamelidis may be used. Surgical treatment is seldom required at this age.

Anal Fissure

This is likely to come about from tearing of the mucosa during the passage of hard scybala. The fissure is often so small that it cannot be seen until the anal folds are gently separated, when it appears as a short linear ulcer. The presence of the fissure is likely to cause spasm of the anus, which increases the constipation and so sets up a vicious circle. Defæcation is painful, and the infant may try and resist the act by arching his back and compressing his buttocks together. A history of this should always lead to a careful examination for a fissure.

Treatment consists of softening the stools by giving paraffin, while the anus may be smeared with an ointment of 2 per cent. novocain. The fissure may then be touched with a stick of silver nitrate.

HERNIA

Inguinal Hernia

This is the most common variety in childhood. Of 1,018 instances of hernia at The Hospital for Sick Children,[1] 773 were inguinal. Boys are much more frequently affected than girls, roughly in the proportion of 9 to 1, and in about a fifth of the cases the hernia is bilateral. Various factors contribute to the production of inguinal hernia. Undoubtedly a congenital weakness at the internal abdominal ring is usually present, and then any sudden or continued strain, such for instance as a sudden bout of coughing, constant crying or vomiting, constipation, or the straining due to a pin-hole urinary meatus, may be enough to produce a rupture. The hernia may appear either in the groin or scrotum, and is often only to be found actually during a bout of coughing or crying. As a rule the tumour can be easily reduced into the abdomen, the gurgling feeling which accompanies its replacement being diagnostic of the condition. The presence of a hernia seems to cause the infant more than mere discomfort, for almost always when the rupture is down he is restless and crying. Inguinal hernia must be distinguished from an encysted hydrocele, which has both an upper and lower limit and is irreducible, and from an undescended testicle, which appears as a small circumscribed tumour that cannot be returned into the abdomen, but may sometimes be coaxed down towards the scrotum, and of course the scrotum on that side is empty.

During the first year inguinal hernia is capable of spontaneous cure, provided that it is prevented from coming down by the use of a suitable truss. In infancy a rubber horseshoe-shaped truss is most suitable, while as the toddling stage is approached a rubber-covered spring truss should be worn, the truss being changed every two or three months as the child grows. Open operation should, if possible, be delayed until the child has learned clean habits, which incidentally will also allow time enough to see whether a spontaneous cure is likely.

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[1] **Paterson, D. H., and Gray, G. M.,** ***Arch. Dis. Child.*****, 1927, 2, 328.**

When an inguinal hernia is produced after a particularly severe straining it may become swollen, tender, and irreducible. In that case the child should be put to bed with the pelvis raised above the level of the head, and then after a few hours gentle pressure will usually enable the bowel to be returned into the abdomen. If, however, this cannot be done, or if vomiting and complete constipation occur to indicate strangulation, immediate surgical relief is required.

Femoral Hernia

This form of hernia is very rare in children, but, as in adults, there is a preponderance of cases in the female sex. Treatment is surgical.

Umbilical Hernia

This is a common form of hernia in infancy, and is due to weakness of the umbilical scar. Any causes of straining which give rise to an increase of the intra-abdominal tension are factors in the production of umbilical hernia. The sexes are equally affected. The hernia appears as a soft swelling, pouting out from the umbilicus, and is easily reducible. In addition there is always some degree of separation of the recti muscles.

A spontaneous cure may be expected in about half the cases, cure generally coming about before the end of the first year, but may take place even up to three years of age, and therefore surgery should, if possible, be delayed until this age. Meanwhile a flat pad of lint should be placed over the umbilicus after the rupture has been reduced, and may be held in place by broad bands of strapping. The recti muscles should be pinched together when the strapping is applied, so that when the skin is released the strapping will keep the pad firmly against the umbilicus. Conical pads which dip into the umbilicus should not be used, as although they prevent the rupture from recurring they at the same time prevent the umbilicus from closing.

Diaphragmatic Hernia

The protrusion of the abdominal contents upwards through the diaphragm is usually due to congenital defects, although it may follow upon traumatic rupture of the diaphragm. As a rule the hernia takes place through the left dome, the aperture being often large enough to allow most of the intestine to pass into the thorax.

Symptoms usually arise soon after birth, but may be delayed for some years. There may be attacks of violent flatulence; and sudden effortless vomiting, often without loss of appetite, may be a persistent and troublesome symptom, accounting for a failure to gain weight. Sudden attacks of dyspnœa and cyanosis are likely to occur, and are due either to pressure on the lungs or to displacement of the heart. During these attacks strange physical signs may be found over the chest. The percussion note may be either tympanitic or diminished in resonance, and breath sounds are likely to be absent, but splashing and gurgling sounds may be heard. Other conditions which may give rise to cyanotic attacks must be excluded, such as congenital heart disease or pulmonary atelectasis. The diagnosis is greatly facilitated by X-raying the child after giving a bismuth meal, which reveals the displacement into the thorax of the stomach or intestine. Between attacks, when the intestines have returned into the abdomen, examination may be negative.

For the cure of the condition surgical operation is needed, but unfortunately the opening in the diaphragm is often extensive, and the operation to close it is a severe one. Surgical treatment should therefore be postponed until three or four years of age unless the suffocative attacks are frequent and severe, or unless symptoms of intestinal obstruction arise.

THE PERITONEUM

Congenital Deformities of the Peritoneum

Congenital deformities of the peritoneum are important in as much as they allow the bowel an undue amount of mobility, and so render it more liable to kinks and twists, with consequent obstructive symptoms. In one variety the mesentery of the cæcum and ascending colon persists in its fœtal condition and is in continuity with the mesentery of the small intestine. Not infrequently this is combined with an incomplete attachment to the posterior abdominal wall, so that the mesentery of the small intestine and ascending colon may simply be tethered at its upper end at the left side of the second lumbar vertebra. Mesenteric deformities are more common than is generally realised. The conditions described above were found by the writer in 6 out of 360 autopsies on children under twelve years of age. The age at which death occurred in these 6 cases is noteworthy; 2 died when three weeks old, 1 at three months, 1 at five months, and 2 at six months. During the period when these 6 cases came to autopsy, 170 infants under six months of age died from other causes, so that mesenteric deformities occurred at post-mortem examination in 3·5 per cent. of infants under six months.

The symptoms have been described by Waugh.[1] Owing to the unusual mobility of the intestine, symptoms of partial or complete volvulus may appear at any time, and are likely to be mistaken for appendicitis. The attacks may be repeated several times until eventually one particularly severe one leads to operation. Careful abdominal examination during an attack may, however, show that the right side of the abdomen looks flattened and feels empty, while the left half or perhaps the upper half may be correspondingly distended, the asymmetrical fulness being due to the mobile intestine becoming bunched up in one quadrant. X-ray examination after a bismuth meal may perhaps show the unusual situation of the cæcum and colon, although by the time the pictures are taken the bowel may of course have returned to its normal position.

Pneumococcal Peritonitis

Pneumococcal peritonitis is more frequently met with in childhood than at any other age. Girls are affected much more often than boys—of instances in childhood 73 per cent. occurred in girls (Barling).

Pneumococcal peritonitis may be divided into two types, primary and secondary. In the secondary type the peritonitis occurs as a sequel to some pneumococcal lesion elsewhere in the body, particularly as a complication of pneumonia, empyema, or otitis media, or it may be part of a general pneumococcal serositis affecting the pericardium, pleura, and meninges, as well as the peritoneum. Cases that arise in boys are likely to be of this secondary type.

In the primary type the peritonitis is not preceded by any obvious pneumococcal illness elsewhere. McCartney and Fraser[2] have shown that primary pneumococcal peritonitis is practically confined to female children, and point out that the peritonitis begins in the pelvic peritoneum, and that pneumococci can be recovered in these cases from the uterus and Fallopian tubes. They regard the condition as an ascending infection which reaches the peritoneum *viâ* the genital tract. It is possible also that the infection may sometimes be blood borne to the peritoneum, coming from some such site as the throat, in which the pneumococcus is a common inhabitant.

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[1] Waugh, G. E., *Brit. Jour. Surg.*, 1928, **15**, 438.

[2] McCartney, J. E., and Fraser, J., *Brit. Jour. Surg*, 1922, **9**, 479.

Symptoms. The onset is acute, the earliest symptom being pain, which is usually localised to the lower half of the abdomen. The temperature rapidly mounts to 103° or more, and there may be frequent vomiting. One of the most characteristic symptoms is diarrhœa, occurring in about 90 per cent. of cases, and usually continuing for some days. This is a particularly noteworthy feature, since in other forms of peritonitis constipation is the rule. Herpes labialis occasionally appears, and the leucocyte count is, as a rule, raised above 20,000 per c.mm. In the early stages there may be little to make out on abdominal examination, and the degree of rigidity is often much less than in other forms of acute peritonitis. After two or three days the rigidity may increase, the abdomen begins to distend, and there may be signs of free fluid. At this stage the fluid is thin and serous, with perhaps flecks of lymph in it. In the most acute cases signs of toxæmia soon appear, the child becomes restless and delirious, and death may occur after an illness which only lasts two or three days. In others the toxæmia is less severe, and the fluid gradually becomes more and more purulent. Occasionally the infection passes on to a subacute stage and the pus becomes shut off to form an abscess, situated usually in the pelvis.

The onset of the secondary type of pneumococcal peritonitis is more difficult to detect, since it is likely to be obscured by severe infection elsewhere, although the symptoms are the same as in the primary form.

The mortality is about 50 per cent.

Treatment The most important question to be decided is whether operation shall be undertaken, and, if so, exactly when. The principles to be applied are much the same as those underlying the surgical treatment of empyema, for the best results are likely to be obtained when sufficient time has elapsed for the pus to thicken, and if by other means the patient's strength can be conserved for a day or two there is every advantage in waiting, but to do this means that one must be confident of the diagnosis. The greatest difficulty will arise when, in the early stages, the signs of inflammation are localised towards the pelvis, for unless the possibility of appendicitis can be excluded there is obvious danger in waiting. The most satisfactory cases are those in which the pus has localised into an abscess. There can be few conditions in which close co-operation between physician and surgeon is more desirable.

The general treatment is of great importance. The child should be nursed in the Fowler position. Sips of water should be given by mouth, but fluids must also be given by other routes. Normal saline may be given per rectum or subcutaneously, or even better by a continuous drip method intravenously (p. 100). Sulphapyridine (M & B 693) should be given in full doses, indeed, if it is given early before much fluid has collected, operation may be avoided. Should the drug give rise to vomiting, it can be given together with the intravenous saline. The relief of pain is best effected by warm fomentations to the abdomen, combined with small doses of opium. A small blood transfusion is often of value, and if it leads to improvement it may be repeated.

Streptococcal Peritonitis

In its mode of infection, symptomatology, and treatment, streptococcal peritonitis is closely akin to the pneumococcal variety, but its incidence is not more than half that of the pneumococcal form. Most of the cases occur secondarily to streptococcal lesions elsewhere, but of 12 cases recorded by Barrington-Ward 5 were regarded as primary, and of these 4 were in girls. It has already been said that the symptoms are similar to those of the pneumococcal cases, but fulminating instances occasionally arise in which diagnosis may be almost impossible. This was so in the case of a girl four years old who was apparently in perfect health until 5 a.m. on the day of her death. She then vomited, and throughout the day lay listlessly, refusing all food ; at 6 p.m. she had a convulsion, and died an hour later. Autopsy showed a slight distension of the intestine and an intense fiery red injection of the whole peritoneum, the peritoneal cavity containing about a pint of thin serous fluid which on culture grew a pure growth of streptococci. The ovaries and Fallopian tubes were extremely congested, and pure cultures of streptococci were obtained from them.

The outlook is even worse than in pneumococcal cases, although the mortality has improved since the introduction of the sulphanilamides ; but these drugs must be given in full doses, half measures will not suffice. Should pus collect, an operation to drain it may be required in a few days.

Gonococcal Peritonitis

This is a very rare form of peritonitis in childhood. It is almost confined to the female sex, and is preceded by gonococcal vulvo-vaginitis. Baer[1] found that out of 477 children with epidemic gonorrhœa, peritonitis only occurred in 5, and one of these proved fatal.

The onset is as a rule sudden, with fever, chills, vomiting, and severe pain in the lower abdomen. At first the temperature is likely to be high, but the condition gradually settles down to become subacute.

If the diagnosis can be made with certainty, conservative treatment should be adopted, since the condition tends to become subacute. The child should be isolated, and nursed in the Fowler position, pain being relieved by the local application of heat. Sulphapyridine should be given by mouth in full doses, in addition to local measures to clear up the vulvo-vaginitis.

Tumours of the Peritoneum

Tumours growing from the peritoneum are rare. Occasionally a sarcoma arises from the peritoneal tissues, and spreads rapidly to form a large mass which resembles clinically a retroperitoneal sarcoma. Cysts of the peritoneum may also arise from the omentum or mesentery, and may form large tumours. Their treatment is surgical.

Retroperitoneal Sarcoma

This is fortunately a rare condition ; it occurred in 5 out of 400 consecutive autopsies at Great Ormond Street. It is most likely to be met with during the first three years, and may affect either sex.

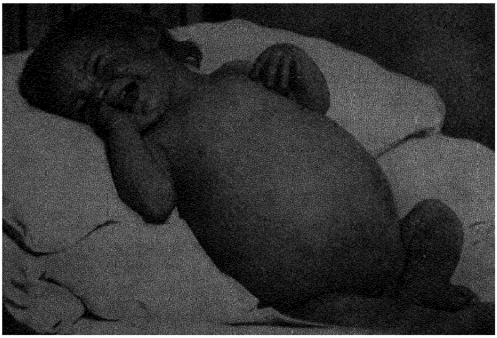

FIG. 37. Retroperitoneal sarcoma in a baby aged one year, showing the abdominal distension.

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[1] Baer, J. L., *Jour. Infect. Dis.*. 1904, 1, 313.

It is probable that more than one form of new growth is included under the term retroperitoneal sarcoma, for the tumours vary considerably in their appearance. Some are for the most part white and soft, but with plum coloured areas of hæmorrhagic necrosis, and have the histological characters of a sarcoma. Others are lumpy, and on section have a whorled appearance with perhaps softer patches of a yellowish colour, and show on section a variety of cells, including fibrous tissue and muscle bundles. These are to be regarded as malignant teratomata. Retroperitoneal sarcomata enlarge rapidly and invade surrounding structures such as the lymph glands, pancreas, liver and kidneys. One such instance was a baby of six months, in whom a tumour had invaded the cortex of the kidney, and during life sarcoma cells were obtained from the urine. Secondary deposits are particularly likely to be found in the mediastinum or in the lungs.

Often the first thing to be noticed is enlargement of the abdomen, soon followed by loss of weight. Frequently there seems to be considerable pain, and the infant may keep up a pitiable whining most of the day. Examination of the distended abdomen shows a hard irregular mass situated as a rule in the midline, and fixed to the posterior abdominal wall. There may be ascites, and œdema of the legs may occur from compression of the inferior vena cava. The hardness and fixity should distinguish these tumours from a mass of tuberculous glands, while the midline position and lack of bulging of one flank helps to distinguish them from renal tumours. Occasionally vast accumulations of scybala may mimic a retroperitoneal growth, but their position is less fixed, and the effect of repeated enemata will soon dispel any doubts.

The course is a rapidly downhill one, death ensuing within a month or so of the initial symptoms.

INTESTINAL WORMS

Thread-Worms (Oxyuris Vermicularis)

This is the most common type of intestinal parasite in childhood. The worms are small, measuring ½ to 1 cm., the female being about twice as big as the male. They are white in colour and are usually described as appearing like little pieces of white cotton. They chiefly inhabit the upper part of the large intestine, especially the cæcum and appendix, whence they migrate down the bowel, and may actually wriggle out of the anus, and set up irritation in neighbouring parts. Thread-worms exist in large numbers in the host, and the infection is often very persistent. Re-infection comes about by the child rubbing and scratching himself to allay the peri-anal irritation, and in so doing he infects his fingers and nails with the ova of the parasites, which are then transferred to food, or directly to the mouth by the child sucking his fingers or biting his nails. The infection is very easily spread from child to child, and in a family of children it is seldom that the infection is confined to one member. Whether an ovum can develop into a mature worm without having to leave the large bowel is uncertain, although Still has advanced good reasons for thinking that this may be so, pointing out that young thread-worms are practically never found in the small intestine, while they may be present in the appendix and large intestine.

Symptoms. Although thread-worm infection is blamed for a great variety of symptoms, it is often difficult to be sure that the symptoms are caused solely by the worms. Thus loss of appetite, under-nourishment, attacks of pallor, headaches, and such nervous manifestations as teeth grinding, night terrors, and even convulsions, are often put down to thread-worms, although it is probable that these symptoms are as much due to the catarrhal condition of the bowel, which is so often present, as to the worms themselves. It is almost invariably the case that the stools which contain thread-worms also contain a great deal of " slime " or mucus, in fact it may be said that thread-worm infection in an otherwise healthy child is generally a fleeting affair and is easily remedied, and that many of the symptoms attributed to thread-worms are really the outcome of long-standing intestinal indigestion. It follows that if treatment is to be successful attention must be paid to the health of the bowel as well as to the eradication of the worms.

There are, however, certain symptoms for which the worms must be held responsible. It has already been pointed out that the worms may escape from the anus and set up a local irritation, and this is particularly likely to happen at night when the child is warm in bed, and may lead to much restlessness and broken sleep. In girls the irritation may spread to the vulva, and the efforts to allay the itching may lead to vulvitis with some discharge. Nocturnal enuresis is also often associated with thread-worms, and it is probable that the perineal irritation is here a contributory factor. In severe cases there may be a good deal of itching during the day as well as at night. Occasionally thread-worms may give rise to recurrent attacks of appendicular colic, which may lead to appendicectomy. The appendix in these cases is swollen, and on opening it the inner surface is coated with mucus in which are found many small thread-worms. The catarrhal condition of the appendix is simply a part of the general catarrh of the large intestine, an appendix in this condition offering a most favourable nidus for the lodgment and development of the worms.

Treatment. With regard to improving the health of the intestine, what has already been said in the treatment of chronic indigestion applies to these cases (see p. 199). Proper management must include a regular amount of exercise each day, regular meals, and adequate rest. A long course of a rhubarb and soda mixture is of value. It must be remembered that both the general and dietetic management of a child with persistent thread-worm infection is at least as important as the use of vermifuge drugs.

Various vermifuges have proved useful. Santonin may be given at night for three consecutive nights, the course being repeated if necessary after a fortnight's interval. It may be prescribed in the following powder :—

℞. Santonini gr. ii.
Pulveris scammoniæ compositi gr. ii.
Hydrargyri subchloridi gr. ss.

Small repeated doses of calomel such as gr. $\frac{1}{6}$ ter die are occasionally helpful. "Butolan"[1] (a carbaminic ester) is often effective ; it is prepared in tablet form, each tablet containing gr. 7½ ; half a tablet should be given twice a day for a week, and should be combined with a morning saline such as magnesia. If the drug is to be repeated, an interval of a fortnight should elapse before starting another course.

Treatment by enemata, given at bedtime, is a popular but usually ineffective remedy. Infusion of quassia may be used, or a simple salt-and-water enema containing a dessertspoonful of salt to a pint of water. Often only five or six ounces is given, but if it is intended to wash out the colon—and remembering how often the appendix harbours the worms anything less than a high colon lavage would seem useless—as much as sixteen to twenty ounces should be given. There is no doubt that an occasional enema is of real value in combating nocturnal irritation, but their regular use, such as every day or every other day, is unwise. Repeated enemata render the anal sphincter patulous, they leave the colon unemployed, while their psychological effect is nothing but detrimental.

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[1] Butolan is also sold under the trade names Diphenan and Oxylan.

Lastly, steps must be taken to prevent the child from reinfecting himself. He should sleep in a sleeping-suit or pyjamas, and as an additional precaution against getting the fingers and nails infected a clean pair of cotton gloves should be worn at night. These can be boiled each morning. The hands should be thoroughly washed and the nails scrubbed before each meal. The perineal irritation can also be checked by smearing a ring of mercury ointment over the anus when the child goes to bed, for the worms seldom pass this barrier.

Whip-Worms (Trichocephalus Dispar)

These worms are occasionally found in company with threadworm infection, and are recognised by the passage of the ova in the stools. The symptoms do not differ from those produced by threadworms, and the treatment is identical.

Round-Worms (Ascaris Lumbricoides)

Round-worms are less frequent than thread-worms. They resemble the ordinary earth worm in appearance, the female measuring about 10 inches and the male about 6 or 7 inches. The ova are passed in large numbers with the stools. As a rule a child only harbours two or three of these worms, but occasionally twenty or thirty are found, and rarely there are hundreds.

As a rule round-worms do not give rise to symptoms, and their presence is unsuspected until one is passed per rectum. They may, however, migrate from their habitat in the small intestine, and may then be vomited, and have been known to pass into the larynx, and to block the bile duct. Fortunately, such migrations are rare. As a rule the worm, when first passed, is alive. The presence of a dead round-worm in the intestine is sometimes associated with severe nervous symptoms which may culminate in convulsions, the symptoms subsiding with the passage of the dead worm.

The most effective drug is santonin, of which 2 gr. may be given in a powder for three consecutive nights. This will bring away any remaining worms, although it often happens that the infection is limited to one worm. After an interval of a week the fæces may be examined for ova, and if these are present the santonin should be repeated.

Tape-Worm (Tænia)

Tape-worm infection is very much less common than either thread-worms or round-worms, and in this country the only tape-worm likely to be met with is tænia saginata, which comes from infected beef. The use of raw meat juice offers a possible source of infection. A full-grown worm may attain a length of 10 feet or more, and as a rule only one worm is present. Tænia saginata may be recognised by the four suckers on its head, which distinguishes it from tænia solium (obtained from infected pork), which has a row of twenty-four hooklets in addition to its four suckers. It is important to be able to recognise the head of the worm, because unless the whole worm, including the head, is passed it may grow again, and treatment will have to be repeated.

A tape-worm does not, as a rule, present any symptoms, and its presence is only detected by the passage of strips of ripe proglottides in the stools. Such strips are generally a few inches long, but may be several feet. Burnet has recorded four instances in which choreic movements were associated with the presence of a tape-worm.

Treatment. The essential point in the treatment is to get rid of the head of the worm, and this calls for a strict *régime.* The child should be in bed throughout the treatment. For two days a fluid diet only should be allowed, and on the evening of the second day a purgative dose of castor oil should be given. On the following morning before food is allowed one teaspoonful of liquid extract of filix mas should be given. This is best administered in four capsule doses of fifteen minims each, given at intervals of a quarter of an hour. If there is difficulty in getting the child to take the capsules, each dose of fifteen minims can be given in a little warm milk or in a teaspoonful of orange juice or syrup. This is followed an hour later by a saline purge, and then in two hours' time an ordinary diet can be resumed.

In order to facilitate the examination of the stools for the head, they should be passed into a chamber lined with black muslin. The stool can then be washed through the muslin, leaving behind the segments of worm. The head will be found by following up the smallest segments. If the head has not been obtained, two or three months should elapse before treatment is re-commenced, in order to allow time for the worm to grow again and give evidence of its continued existence by the passage of fresh segments.

Lambliasis (Lamblia or Giardia Intestinalis)

This is a flagellate protozoon which inhabits the small intestine, and is detected by the presence of the flagellates or their cysts in the stools. As a rule the infection is symptomless, but recurrent diarrhœa with the passage of pale bulky motions, mucus, and undigested food, may occur. Although the infection may persist for years, symptoms, if present at all, tend gradually to disappear.

The most successful drug is Atebrin [1] (Quinacrine), which should be given over a course of three to five days. In addition the general and dietetic measures that have been outlined under chronic indigestion should be instituted, together with a temporary reduction of fat in the diet.

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[1] Atebrin is prepared in 0·1 gm. tablets. The dose is: under 2 years, ¼ tablet twice daily; 2 to 6 years, ½ tablet twice daily; 6 to 9 years, ½ tablet thrice daily; 9 to 12 years, one tablet twice daily.

CHAPTER XI

ABDOMINAL TUBERCULOSIS

This term is used here to embrace tuberculosis of the intestine, mesenteric glands, and peritoneum, but does not include tuberculosis of the other abdominal organs such as the kidneys, suprarenals, spleen, etc.

It is difficult to obtain accurate statistical evidence to show the relative incidence in childhood of abdominal over other forms of tuberculosis, but undoubtedly the incidence of abdominal tuberculosis is on the wane, for while Still [1] found that 88·3 per cent. of tuberculous children had lesions in the abdomen, the writer, in a series of 400 autopsies in children, found tuberculous ulceration or caseation in the abdomen in only 37·5 per cent. of tuberculous children. The latter figure excludes a mere terminal dissemination of miliary tuberculosis over the peritoneum, although this was probably included in Still's figures.

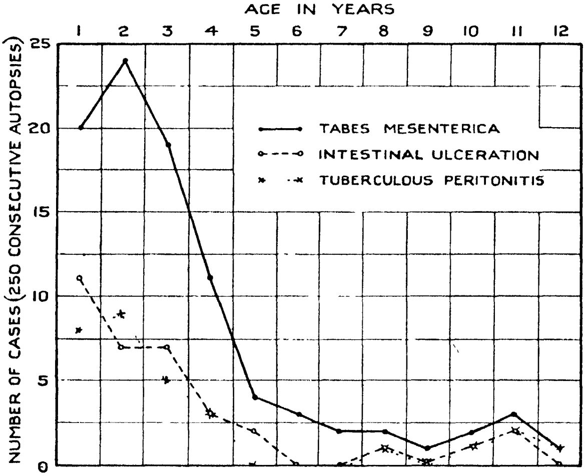

Fig. 38. Chart showing the relative incidence post-mortem of tuberculous enteritis, tabes mesenterica and tuberculous peritonitis.

As in other forms of tuberculosis in childhood, abdominal tuberculosis is most likely to be fatal during the first two or three years. This is shown in the chart (Fig. 38), which is constructed from an analysis of 250 consecutive autopsies in children under twelve years of age. It also shows the separate post-mortem incidence of tuberculous peritonitis, tabes mesenterica, and tuberculous intestinal ulceration, from which it will be seen that of these three types of lesion tabes mesenterica is the most common. Intestinal ulcerations were found in 22 per cent., a figure which agrees closely with that of Blacklock,[1] who found ulcerations in 18 per cent.

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[1] Still, G. F. "Common Disorders and Diseases of Childhood," 1st edit., 1909, p. 375.

The mode of infection is chiefly by the ingestion of tubercle bacilli, which may be of either human or bovine strain. When the infection is due to the bovine type of bacillus the organism is most likely to be swallowed in milk, and recent figures indicate that this is a greater source of danger in Scotland than in England. Milk is, of all foods, by far the most likely to act as a medium for the transport of tubercle bacilli, and cream is, of course, as likely to be infected as the milk from which it is drawn. In 1931 it was found that 6 to 7 per cent. of the specimens of fresh milk in this country contained living tubercle bacilli. Organisms reaching the body in this way may sometimes be caught up in the pharyngeal lymphatic traps and become side-tracked to the cervical glands, while others are swallowed and reach the intestine, where they may produce ulceration of the mucosa or may pass *viâ* the lymphatics to the mesenteric glands, whence they may invade the peritoneum. Human strains of tubercle bacilli may reach the intestine in a similar way by being inhaled on to the pharyngeal wall and then swallowed with the secretions, or, of course, food may be infected with the human strain. Children with a tuberculous lesion in the lungs are also likely to infect their intestine secondarily, by swallowing sputum containing the organism. It is not at all uncommon to find recent tuberculous lesions in the abdomen when a child has died from intrathoracic tuberculosis.

It cannot be too strongly emphasised that milk-borne tuberculosis is a preventable disease, prevention being effected either through the sale of milk which has been efficiently pasteurised before it is retailed, or by boiling the milk in the private house. Tuberculin-tested milk, although free of tubercle bacilli, is produced on too small a scale to exert much effect in lowering the incidence of milk-borne tuberculosis, and, moreover, its cost puts it beyond the reach of all but a few.

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[1] Blacklock, J. W. S.. *Med. Res. Council,* London, 1932, Special Report, No. 172.

Pathology. The pathological events can be divided into three stages :

(1) Tuberculous enteritis.

(2) Tabes mesenterica (tuberculous mesenteric glands).

(3) Tuberculous peritonitis.

Tuberculous Enteritis. There is no doubt that the tubercle bacillus may often penetrate the intestinal mucosa without leaving any visible injury, in fact, of 100 cases of abdominal tuberculosis, lesions of the mucosa were only found in 34. In other cases large areas of the mucosa may be destroyed, while glandular and peritoneal involvement may be by comparison quite slight.

Tuberculous ulceration generally begins at the lower end of the ileum or in the cæcum, the lesion often starting in the Peyer's patches. The ulcers tend to spread in a circular direction round the gut, following the line of the lymphatics. They vary considerably both in number and size, and in advanced cases may coalesce to produce large areas entirely denuded of mucosa. The ulcers diminish in number as the small intestine is traced upwards, and even in the most severe cases the duodenum will not contain more than one or two.

Tabes Mesenterica. The first glands to be affected are usually those at the lower end of the mesentery in the right iliac fossa, draining the lower end of the ileum. One gland, or several, may be involved. As a rule several separate points of caseation first appear in the gland, and these gradually coalesce until the whole gland is caseous. The gland, or glands, may heal by fibrosis, often accompanied by calcification, in which case they can be seen on an X-ray film. In other cases they become adherent to neighbouring coils of intestine, and these adhesions may later on give rise to kinking of the bowel, and lead to attacks of partial or even complete intestinal obstruction, or the glands may liquefy and discharge their contents into the peritoneum, in which case the whole peritoneal surface becomes sown with tubercles, leading to generalised tuberculous peritonitis. If, however, previous adhesions have shut off the glands from the general peritoneal cavity, a local abscess will form instead. It is also possible for a gland to infect the blood stream, and then, according to the dose of the infection, to produce either isolated tuberculomata in other parts of the body or generalised miliary tuberculosis.

Occasionally other glands than those of the mesentery become involved. The para-aortic glands may be affected, as may those that drain the colon

Peritonitis. Infection of the general peritoneal cavity gives rise at first to the formation of miliary tubercles, to which the peritoneum may react by producing a fluid effusion (ascitic variety). The fluid is clear, straw-coloured, sterile, and has a lymphocytic cell reaction. By keeping the two layers of peritoneum apart, the effusion tends to prevent the formation of adhesions, which probably assists the process of healing. In other cases ascites does not occur, and the coils of intestine become matted together, sometimes to such an extent that the peritoneal cavity is practically obliterated (plastic variety). The adhesions are often so dense that at post-mortem examination it is impossible to separate the intestine, which has to be stripped from the posterior abdominal wall in a conglomerate mass.

Symptoms. For the convenience of description, the clinical manifestations of abdominal tuberculosis may be divided into three headings corresponding to the three pathological divisions just described, namely tuberculous enteritis, tabes mesenterica, and peritonitis, although they should not be regarded as isolated events, since they are in reality parts of a continuous process, indeed all three stages may be co-existing, and the physician must be prepared to find the symptoms of one stage overlapping those of the others.

Tuberculous Enteritis. Tuberculous ulceration of the intestine has generally an insidious onset, beginning with attacks of diarrhœa which become more frequent in spite of treatment. The diarrhœa is seldom intense, but is usually persistent. The stools are loose, slightly offensive, containing shreds of undigested food and mucus, and may be repeatedly streaked with blood, and bacteriological examination may show the presence of tubercle bacilli. The diarrhœa is accompanied by progressive wasting, which may be so severe and rapid as to seem out of proportion to the other symptoms, and to be more than can be accounted for merely by tuberculous toxæmia. An equally important factor in the wasting is the destruction by ulceration of large areas of that part of the intestinal mucosa normally concerned with the absorption of the products of digestion. The child usually complains of pain in the region of the umbilicus, varying in severity from mere discomfort to sharp colicky attacks. To the local symptoms are added the general manifestations of tuberculosis, such as a temperature of 3 or 4 degrees, flushing of the face, and sweating.

On examination the abdomen is somewhat tumid, and may be tender on palpation. Generally there is enough rigidity to prevent one from feeling structures on the posterior abdominal wall, but it may be possible to make out enlarged mesenteric glands, especially in the right iliac fossa.

Tabes Mesenterica. The symptoms of tabes mesenterica are as a rule less defined than in the case of either tuberculous enteritis or peritonitis, in fact there is often nothing decisive about them. There may be a history of attacks of colicky pain, seldom sufficiently intense to make the child cry, but causing a good deal of discomfort, and interfering with the appetite. Undue tiredness is often a complaint. The weight becomes stationary, or may gradually drop. The state of the bowels varies, but most commonly mild attacks of diarrhœa alternate with constipation. The stools are in many cases normal, but when several glands are caseous the absorption of fat *viâ* the lacteals is interfered with, and the stools may then become large, pale, and offensive, resembling those of cœliac disease. Analysis of the stools bears out this similarity, for in both conditions the tried stool may contain a great excess of split fat. The fatty nature of the stools can be quickly reduced by giving a diet devoid of fat for a few days.

Inspection of the abdomen seldom shows anything unusual, but careful palpation will reveal the presence of enlarged glands on the posterior abdominal wall, usually in the right iliac fossa. The glands vary in size, and may be no bigger than a hazel nut. As a rule they are multiple, firm, slightly movable, and a little tender. They are easily overlooked unless palpation is done with the child lying comfortably and the examiner seated on the child's level. It is useless to expect to feel them if the child is crying. Nevertheless, the other symptoms are often so vague that the diagnosis should never be made until the glands have actually been felt.

It is by no means uncommon to mistake fæcal concretions for enlarged mesenteric glands. Occasionally it may be possible to determine that the fæcal masses can be indented during palpation, and they are likely to be more movable than glands, but even so a real doubt may exist, and it is then better to defer the diagnosis for a week or so in order to allow time for the effect of daily laxatives to be seen. It is as well to begin the evacuation by giving an enema. Any doubts will be resolved if the masses disappear after the bowels have been well opened for a week or so.

Tuberculous glands in the mesentery may become adherent to neighbouring coils of intestine and may then give rise to symptoms of intestinal obstruction, and these symptoms may arise long after the original tuberculous infection has taken place. Occasionally the obstruction is complete and calls for immediate operation, but more often the symptoms are those of partial obstruction, the attacks recurring at intervals. The history is then likely to be one of bouts of constipation accompanied by gradually increasing abdominal distension, and culminating after perhaps a fortnight or so in several copious and rather offensive vomits. As the attack passes off the abdominal distension subsides and the child is a great deal more comfortable, but the whole sequence of events is likely to be repeated.

Tuberculous Peritonitis. Tuberculous peritonitis has generally an insidious onset, with a history for some weeks of abdominal discomfort or pain, anæmia, diarrhœa, and loss of weight. The clinical picture can be divided into two types—the ascitic form and the plastic form.

In the ascitic variety the first thing to be noticed may be progressive enlargement of the abdomen. The temperature is generally raised 2 or 3 degrees. Examination shows the abdomen to be distended, particularly in the flanks, and on percussion the presence of shifting dulness indicates an accumulation of free fluid. Occasionally the fluid may be loculated by peritoneal adhesions, and the area of dulness will then be circumscribed, and not affected by changes in the child's position.

The course taken by the ascitic form varies. In some cases the appetite is maintained and the nutrition remains tolerably good, and after some months the fluid gradually absorbs and the child makes a slow recovery. In other cases the ascites is quickly absorbed, but the abdomen remains distended and takes on the features of the plastic variety.

Plastic tuberculous peritonitis is more common than the ascitic form, to which it may be a sequel, and on the whole the clinical picture is a more striking one. The child with plastic tuberculous peritonitis looks miserably ill. The appetite is generally bad, vomiting is frequent, and the bowels are loose. The general symptoms of tuberculous infection are well marked, and include a temperature of 102° F. or higher, flushing of the face, rapid loss of flesh, and sometimes sweating. On examination the abdomen is swollen, and is in marked contrast to the wasting of the rest of the body. On palpation it has a characteristic "doughy" or packed feeling, that is to say, although not really rigid it is impossible to make out any definite structures in the abdomen, because the examining fingers are everywhere resisted. The abdomen feels as though it were tightly packed with cotton wool. Although it is seldom possible to feel anything inside such an abdomen, one can sometimes make out the rolled-up tuberculous omentum lying as a sausage-shaped tumour across the epigastrium. Such a tumour can be distinguished from the liver by the fact that it has an upper as well as a lower border, nor is it likely to be mistaken for the tumour of a chronic intussusception, since the history is quite different, and the rolled-up omentum does not undergo periodic hardening and softening.

Plastic tuberculous peritonitis may also develop locally around a tuberculous intestinal ulcer or in the neighbourhood of a caseous mesenteric gland. Localised peritonitis of this sort is most commonly situated in the neighbourhood of the cæcum and lower end of the small intestine, where it may give rise to a tender mass in the right iliac fossa. The gradual history, with neither vomiting nor constipation as prominent symptoms, and the negative findings on rectal examination, help to distinguish such a mass from an appendix abscess.

The course of plastic peritonitis tends to be a downhill one, especially in young children. The distension gradually increases, the umbilicus may become opened out, and the anterior abdominal wall in this region may become adherent to the underlying mass of matted bowel. Occasionally an abscess slowly forms at the umbilicus and eventually ruptures, to discharge much caseous matter. In some cases a fæcal fistula forms as well, and is due to the rupture of a tuberculous ulcer into an area of peritoneum which has already become walled off by adhesions from the general peritoneal cavity. Although a child in such straits is dangerously ill, recovery is yet possible. With the discharge of caseous material the abdominal distension lessens, and a chronic sinus remains, which after several months of fitful discharge eventually closes.

Diagnosis. In addition to the general symptoms of tuberculous infection, the special features which attach to the three forms of abdominal tuberculosis are: in tuberculous enteritis, the persistence of diarrhœa with perhaps occasional streaks of blood in the stools, and rapid emaciation. In tabes mesenterica, attacks of abdominal pain, a failure to thrive, occasionally the passage of fatty stools, and above all the presence of palpable glands in the abdomen. In tuberculous peritonitis, ascites may be present; but the plastic form is more common, in which the distended abdomen has a characteristic doughy or packed feeling, and occasionally a rolled-up omentum can be made out. Wasting is most rapid in tuberculous enteritis and in plastic peritonitis. When there is doubt as to the nature of the abdominal complaint, confirmatory evidence is sometimes to be obtained from the presence of active lesions in the chest.

Additional help in the diagnosis may be obtained from special investigations. Examination of the stools may show tubercle bacilli, and this is especially likely when there is tuberculous ulceration of the intestine. X-ray examination is also of assistance in some cases of tabes mesenterica. If the glands have begun to calcify, the shadows thrown by these glands have a typical stippled appearance owing to the irregularity of the calcification. They must be distinguished from urinary calculi both by their appearance and by their position, for it can generally be determined that they are not actually in the line of the urinary apparatus. In those cases which show the symptoms of partial obstruction owing to adhesions, help may sometimes be had from an X-ray taken after a bismuth meal.

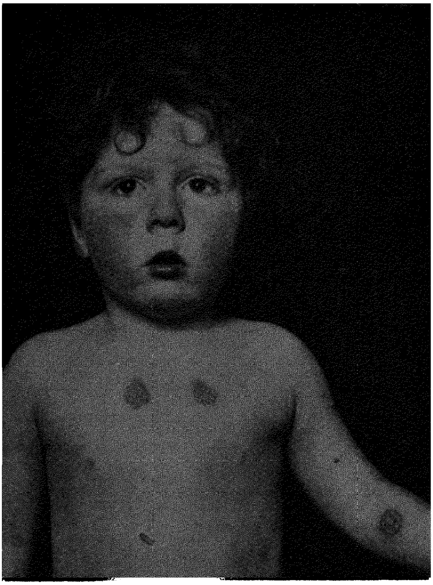
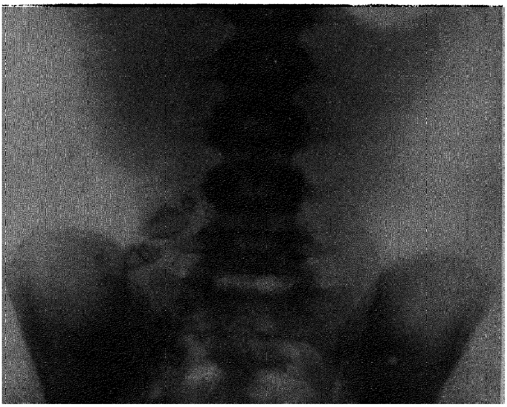


FIG. 39. Boy, aged four years, showing a positive Vollmer patch test on the chest, and a strongly positive Mantoux intradermal reaction on the left forearm.

Tuberculin tests are often of great assistance. Those most commonly employed at the present day are the intradermal test of Mantoux, and the Vollmer patch test. The former consists of the intradermal injection of 0·1 c.c. of 1 in 1,000 or 1 in 10,000 solution of tuberculin. It may be pointed out that although the tuberculin employed in this test is prepared from the human strain of bacillus, a positive reaction is obtained just as readily in a child who is infected with the bovine strain. A positive reaction appears as a red œdematous swelling at the site of inoculation after twenty-four to forty-eight hours, and

PLATE III.



X-ray showing calcifying tuberculous glands in the right iliac fossa. From a girl aged six years who had complained for several months of vague abdominal pains.

may take several days to subside, in fact some staining of the skin may remain for several weeks. A positive reaction may be expected in over 90 per cent. of tuberculous patients.

The patch test has the advantage of avoiding an injection. The patch consists of a strip of adhesive plaster containing three squares of filter paper, the two outer squares being impregnated with tuberculin while the centre one is a control. The patch is applied to the chest or back, after the skin has been cleaned with ether to remove all grease, and removed after forty-eight hours ; twenty-four hours later the test is read. A positive reaction is shown by an erythematous area opposite the outer pieces of filter paper; when strongly positive, minute yellow points may appear on the erythema. The reaction fades in about a week. The patch test is open to more error than the intradermal test ; for example, the test may fail if the skin is imperfectly cleansed, and different batches of testing strips may vary a little in their reliability, but even so the discrepancy between the two methods is less than 10 per cent.

As to the interpretation of these tests, a negative reaction may generally be taken to indicate that the child has not been infected with the tubercle bacillus, but it should be remembered that if the tuberculous process is in an advanced state, or has become generalised, the tests are likely to become negative, and so are sometimes negative in tuberculous peritonitis. The value of a positive reaction is greatest in the first three years of life, but later on its interpretation becomes more difficult, since it may merely indicate that the child has been infected at some time in the past, but it does not necessarily follow that the symptoms under consideration are due to tuberculosis. During the first three years the significance of a positive intradermal tuberculin reaction was found by Monrad to be as follows : during the first year a positive reaction was invariably associated with active tuberculosis ; during the second year 88 per cent. of positive reactors showed clinical signs of active tuberculosis ; and during the third year 81 per cent. showed evidence of active infection.

Differential Diagnosis. Various conditions may be confused with abdominal tuberculosis. In infants persistent diarrhœa with wasting and failure to thrive is more often the outcome of improper feeding than the result of tuberculosis. In older children the symptoms of chronic indigestion may lead to confusion, but the long period of time for which the symptoms have been present is often enough to make the diagnosis of tuberculosis unlikely. Abdominal distension and severe wasting are features of cœliac disease, and the light fatty stools which characterise this disease may sometimes be met with in widespread tabes mesenterica, but the abdomen in cœliac disease is soft and easily palpated and is therefore quite unlike that of tuberculous peritonitis, while the palpation of enlarged glands in tabes mesenterica will distinguish that condition from the cœliac affection. The differential diagnosis between fæcal accumulations and tuberculous glands has already been mentioned. The recurrent attacks of partial obstruction due to tuberculous adhesions may simulate bouts of cyclical vomiting, but in the latter condition the vomiting is an early symptom and is accompanied by severe ketosis, but not by abdominal distension. The differentiation between a chronic intussusception and a rolled-up omentum, and between an appendix abscess and a tuberculous mass in the region of the cæcum have already been considered.

Recently Crohn[1] has described a condition of chronic inflammation of the lower end of the ileum (regional ileitis), leading to thickening and rigidity of the bowel, sometimes associated with local adhesions and the formation of fistulæ, and eventually reducing the size of the lumen. The disease is very likely to be mistaken for plastic tuberculosis. The symptoms include abdominal pain, alternating diarrhœa and constipation, and loss of weight, to which may be added evidence of intestinal obstruction such as abdominal distension and visible peristalsis of the small intestine. Relief is afforded by a short-circuiting operation or by resection of the affected area.

Prognosis. The outlook varies with the particular type of abdominal tuberculosis, and also with the age. In general terms the outlook is grave when the disease occurs under two years of age, but improves steadily as the child gets older. The presence of tuberculosis in other parts, especially in the lungs, affects the prognosis adversely.

The most hopeful cases are those of tabes mesenterica. Under careful and prolonged treatment even large glandular masses may gradually subside, until after a matter of several months, or more usually years, the child can be pronounced cured. The possibility, however, of a general infection *viâ* the blood stream is always present so long as there is active disease in the glands, and the likelihood of this accident is greatest in the early years. On this account the prognosis should always be a guarded one. When tuberculous peritonitis has arisen, the outlook is a little better in the ascitic variety, although it may happen that the fluid is soon absorbed and the case changes to the plastic type, but even so the abdomen is capable of remarkable recovery, and it may be said that except during the early years a hopeful attitude is usually justified.

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[1] Crohn, B. B. *et alia. Jour. Amer. Med. Assoc.*, 1932, **99**, 1323.

Treatment. The two most important aspects in the treatment of abdominal tuberculosis are rest and fresh air. If the child can be nursed at the seaside, so much the better, and in this country the various resorts on the Kentish coast are most suitable, while for those who can afford it the sunshine and fresh air of the Swiss Alps is excellent. Except in the most severe cases with a swinging temperature, open air treatment should be employed, and the child should lie out of doors for most of the day. During the warm months the trunk and limbs should be exposed to the sun and air for gradually increasing periods, and even in the colder months short exposures to the midday sun in a corner sheltered from the wind are beneficial. The effect of sunlight and fresh air playing on the skin is much to be preferred to exposure to artificial ultra-violet light, although if the child cannot be taken to the seaside or the country artificial sunlight should be used, but its effect on the general health needs to be very carefully watched, and over-exposure at the beginning of treatment must be particularly avoided. Ultra-violet light is likely to do more harm than good when the temperature shows a big daily swing. Rest is perhaps even more important than fresh air, and the rest needs to be thorough. Skilled nursing is required, as the child should do nothing for himself, since anything less than this implies inadequate rest. The child should lie on his back all day on a bed or spinal chair which can be wheeled on to a balcony. The clothing needs to be warm, but the child should not be so muffled up that only the face can be seen, since this removes much of the benefit of the open air treatment. Gentle massage to the limbs helps to maintain their tone, and is a useful adjunct.

Rest should be complete until the temperature has settled, and the weight is rising. At this stage the child may begin to get up, but convalescence must be gradual, and will extend over several months. It is at this stage, too, that estimations of the erythrocyte sedimentation rate (see p. 393), repeated every few weeks, offer a valuable guide as to the amount the child may do, for the sedimentation rate should be kept within the normal limit of 10 mm. in one hour.

The diet should be of plain fare, but nourishing, and served attractively. " Roughage " should be avoided, especially when there is any diarrhœa, while if there is tabes mesenterica with the passage of pale fatty motions, the fat in the diet must be curtailed and the milk should be skimmed. Otherwise the diet should be made up of fresh boiled milk, milk puddings, butter, eggs, white fish, meat broths, pounded chicken or veal, a little well-boiled potato and sieved green vegetables, and well-stewed fruit. Cream may be given, provided that it comes from a reliable source and does not interfere with the appetite.

As to medicines, healing by calcification should be promoted by giving calcium gluconate 10 gr. three times a day, or syrup of calcium lactophosphate a teaspoonful thrice daily, and combining this with vitamin D in the form of cod-liver oil a teaspoonful twice daily, or one of the concentrates such as Adexolin or Radiostoleum ten drops twice daily. When diarrhœa is troublesome, small doses of castor oil should be tried, combined with opium, as in the following prescription :—

℞. Olei ricini m. v.
Tincturæ opii m. ss.
Mucilaginis acaciæ q.s.
Aquam menthæ piperatæ ad ℥i.
One teaspoonful thrice daily.

for a child of one to two years.

Inunctions of cod-liver oil, mercury, and iodoform into the abdominal wall have been widely recommended, and there is no doubt that drugs can be absorbed in this way, but it seems paradoxical to advise complete rest and at the same time to massage the abdomen, for surely if rest is essential for the healing of a tuberculous lesion this is hardly to be obtained by massaging the site of the disease. Moreover, there is no good reason to suppose that a drug rubbed into the anterior abdominal wall is any more likely to reach glands situated on the posterior abdominal wall than would inunctions over other parts.

Treatment by injections of tuberculin has had a considerable trial, although in the few cases in which the author has used it it has seemed to make but little difference. It is important to employ small doses, beginning with 1/100,000 mgm., and if this is not followed by any reaction to repeat the injection at ten day intervals.

Lastly comes the question of surgical treatment. Although good results have been recorded in the ascitic form of tuberculous peritonitis by allowing the ascitic fluid to escape, figures show that such a result is not to be relied upon, indeed it is likely that the mere presence of the fluid has a value in keeping the layers of peritoneum apart, so tending to prevent the formation of adhesions. Drainage of the fluid should not be resorted to until medical treatment has had a thorough trial over two or three months. When the peritonitis is of the plastic variety laparotomy is a most undesirable adventure, for it is often impossible to tell when the peritoneal cavity has been reached, but it must sometimes be undertaken when acute obstruction arises from tuberculous bands and adhesions.

Prevention. When a child already has pulmonary tuberculosis it is practically impossible to prevent infected sputum reaching the abdomen. Steps can, however, be taken to prevent abdominal infection from tuberculous milk, and these have already been indicated at the beginning of this chapter. Prevention can be carried out either by using only tuberculin-tested milk, or, more practicably, by the sterilisation of milk by pasteurisation or by boiling. Unless tuberculin-tested milk is used, all milk given to children should be rendered safe by one of these two methods of sterilisation. Legislation to compel the pasteurisation of milk before it is retailed would go far towards stamping out milk-borne tuberculosis. At the present day much of the milk sold in our big cities is pasteurised, with the result that milk-borne tuberculosis has become mainly a disease of country-bred children, where the milk consumed is for the most part unsterilised.

One is sometimes asked up to what age children should be given sterilised milk. The answer should be throughout childhood, for the sterilisation of milk in no way impairs its nutritive value, and the rest of the diet will contain sufficient vitamin C to counteract the small amount of this vitamin destroyed in the sterilisation of milk. It has been suggested that small doses of tubercle bacilli may enable the child to acquire immunity to the disease, in fact this is the basis of prevention by B.C.G. vaccine—an oral vaccine of tubercle bacilli which have previously been rendered avirulent. But to apply this argument to the giving of unboiled and possibly infected milk is thoroughly mischievous, for not only is it impossible to regulate the dose of tubercle bacilli given in this way, but the organisms are virulent, and furthermore the chance of a generalised dissemination of tuberculosis from a small focus of infection is greatest in the early years, at a time when milk forms the bulk of the diet.

CHAPTER XII

DISEASES OF THE LIVER, GALL BLADDER, AND PANCREAS

DISEASES OF THE LIVER

Jaundice

Jaundice in the Newborn. Of the various causes of jaundice during the first month of life, simple or physiological jaundice (p. 24), icterus gravis (p. 25), infective jaundice (p. 27), syphilitic jaundice (p. 651), and acholuric familial jaundice (p. 443) are described in other chapters. There remains jaundice occurring in association with congenital obliteration of the bile ducts.

Jaundice in Association with Congenital Obliteration of the Bile Ducts

In this condition jaundice appears after a week or ten days and gradually deepens, the illness eventually terminating fatally after dragging on for perhaps several months. The condition has occasionally been recorded in more than one member of a family. Boys are more likely to be affected than girls. Syphilis is not a cause, although an instance of syphilitic stenosis of the bile duct in a newborn child has been reported.

Symptoms. The infant usually seems healthy enough at birth, and jaundice seldom appears before the second week ; to begin with it may fluctuate in degree, but the tendency is for it to get progressively worse. The stools soon become clay-coloured, the urine becomes dark brown with bile pigment, the liver gradually enlarges and becomes increasingly firm, and the spleen is also palpable and may reach as far as the umbilicus. Hæmorrhages may occur into the skin or from the stomach and intestines. The general condition remains tolerably good for some weeks, but gradually the weight drops, and finally the infant becomes comatose, and dies at some time between three and eight months of age.

Pathology. The liver is always affected, and shows a condition of biliary cirrhosis. It is enlarged, deeply jaundiced, and has a slightly granular surface interlaced with fine white scars. Microscopically the distribution of fibrous tissue is unilobular, and among the strands of fibrous tissue are to be found the remains of obliterated bile ducts and often numerous young bile canaliculi. The liver cells contain a heavy deposit of bile pigment. The obliteration of the bile ducts may affect any portion of the extra-hepatic biliary system. The gall bladder may be shrivelled and even difficult to recognise, and a part or the whole of the common bile duct may be represented by an impervious fibrous cord. The obliteration is easily demonstrated at autopsy by opening the duodenum and pressing along the line of the bile passages. In the normal infant bile can be expressed in this way into the duodenum. Occasionally biliary cirrhosis occurs in infants in whom the extra-hepatic bile passages are patent, although the symptoms do not differ from those given above. It is probable that in these cases there is an obliterative cholangitis confined to the intra-hepatic biliary system.

The explanation is uncertain. In his original description of the condition Thomson held that the cirrhosis was secondary to the obliteration of the bile ducts, while Rolleston looks upon the cirrhosis as the primary condition, brought about by toxins derived from the mother during pregnancy and injuring the fœtal liver, and giving rise to a descending obliterative cholangitis.

Diagnosis. This form of jaundice can be distinguished from physiological jaundice by its later onset, its progressive deepening, and by the paleness of the stools. Icterus gravis appears earlier, and is a more urgent condition, and is accompanied by an erythroblastæmia. The abnormal fragility of the red cells will help to distinguish cases of acholuric jaundice.

The outlook is hopeless. Treatment is of no avail.

Catarrhal Jaundice

This is the most common form of jaundice after the neonatal period. The disease reaches its highest incidence between four and ten years of age. The sexes are equally affected. Although the condition may be met with at any time of the year, there is a maximum seasonal incidence in the autumn months.

Catarrhal jaundice is now recognised to be of infective origin, although the nature of the infecting agent is unknown, cultures of the blood, stools and urine failing to show any pathogenic organism. It is most probable that the infection is air-borne, spreading from case to case by droplets of sputum. The incubation period lies between three and five weeks, and so it often happens that several children exposed to a common source of infection develop the disease within a few days of each other, giving a false impression that the incubation period is a matter of only a few days. The illness often occurs in small but long drawn out epidemics in schools and among nursing staffs, and may affect several members of a family.

Symptoms. There is usually a history of gastro-intestinal disturbance for three or four days before the jaundice makes its appearance. During this stage the child runs a temperature of 2 or 3 degrees and is irritable and disinclined to take food, the tongue becomes furred, and the breath is unpleasant. Vomiting is usual, and may become frequent, and there may be some mild abdominal pain. The bowels are usually constipated, but there may be diarrhœa for a day or so, and the stools may be offensive. After three or four days the temperature falls, and the motions then become white or clay-coloured. Jaundice first appears in the conjunctivæ, and within a day or two the skin becomes jaundiced, the colour varying from a pale lemon to a deep yellow. Bile pigments appear in the urine shortly after the eyes are discoloured, the urine becoming brown and staining the clothes. With the appearance of jaundice the pulse tends to become slow, and the skin may itch, although this is not so often complained of by children as by adults. If the child is examined in this stage the liver is found enlarged, reaching two or three fingersbreadth below the costal margin, and it may be slightly tender. The spleen is sometimes palpable, but not always so ; it seldom extends more than one or two fingersbreadth below the costal margin, and is not tender. The Van den Bergh test gives a biphasic reaction, indicating that the jaundice is partly obstructive and partly due to disordered action of the liver cells, and the bilirubin level in the serum may rise to as much as ten times the normal.

Pathology. The opportunity of studying the liver seldom arises. The condition is regarded as infective, the illness beginning with a duodenitis, whence the infection ascends the bile passages, causing an obstructive cholangitis and eventually involving the liver. It is however possible that the infection may be blood-borne to the liver and thence descend the bile passages.

Course and Prognosis. The outlook is almost invariably good. The jaundice begins to fade after about a week and recovery is generally complete within a fortnight. There are, however, two possible alternatives which must always make one treat this disease with respect. Occasionally the jaundice deepens while the liver shrinks in size, and the child sinks into a drowsy state to succumb in a few days from acute necrosis of the liver. In others the jaundice persists for several weeks or months, and the liver slowly becomes more and more firm and fibrotic. (Subacute necrosis: biliary cirrhosis.) The later history is then likely to be one of repeated attacks of jaundice, each leaving the liver more damaged.

Treatment. The child should be nursed in bed, even though the jaundice may be of the mildest. As to diet, simple fluids such as lemonade or barley water may be given plentifully, and should be well sweetened. Fat should be entirely eliminated from the diet until the jaundice has completely disappeared. During the first few days Benger's Food made with skimmed milk, or chicken broth, or fruit jellies, may be given, but often semi-solid food in any form is not tolerated, and only a purely fluid diet is possible. As the appetite returns the diet should contain plenty of carbohydrate, honey, jam, toast, fruit and cereal puddings made with skimmed milk being suitable.

Drugs. At the outset it is usual to give a brisk purge, a grain of calomel being the most suitable. Thereafter salines and biliary antiseptics should be given. Magnesium sulphate has a valuable cholagogic effect and may be given in half-drachm doses three times a day. Repeated small doses of calomel (gr. $\frac{1}{6}$ ter die) are useful, or other biliary antiseptics such as bismuth salicylate gr. 10, or hexamine gr. 3, may be prescribed.

Acute Necrosis of the Liver (Acute Yellow Atrophy)

This is fortunately a rare occurrence in childhood. The majority of cases begin as a simple catarrhal jaundice. The condition has occasionally followed the too vigorous arsenical treatment of syphilis, and also occurs in delayed chloroform poisoning. A closely analogous condition arises in poisoning by phosphorus.

Symptoms. The early symptoms will as a rule be those of catarrhal jaundice, and include fever, loss of appetite, and vomiting, followed in a few days by jaundice. Instead of following the usual course of catarrhal jaundice, however, the fever continues, vomiting persists, the jaundice deepens, and hæmorrhages are likely to appear in the skin or may occur from the stomach or bowel. The urine becomes scanty, heavily pigmented with bile, and albuminous, and on microscopical examination may contain crystals of leucine and tyrosine. The liver rapidly diminishes in size and soon cannot be felt, and the area of liver dulness may be reduced to one or two intercostal spaces or may even disappear entirely.

Towards the end the temperature falls below normal, and symptoms of a profound toxic influence on the nervous system appear (cholæmia).[1] The child becomes more and more drowsy, but this may give place to periods when he sits up wailing or screaming, and refusing to be comforted. Local paralyses such as squints or weakness of the palate may appear, and the plantar responses may become extensor. Within a few days the child passes into a terminal state of coma, which may be interrupted by convulsions.

Pathology. Post-mortem examination shows the liver to be considerably shrunken and soft, with its capsule wrinkled. On section, the organ is made up of yellow and red areas, the former containing liver cells in an advanced state of fatty degeneration, while in the latter the destruction of the liver tissue is even more pronounced, and there are large hæmorrhagic extravasations.

Prognosis. Cases that run an acute course such as that described above are almost invariably fatal after an illness of two or three weeks. Occasionally the course is less acute, and then after several weeks of grave illness a slow recovery may be made, but is likely to be followed by recurrent attacks of jaundice with progressive fibrosis of the liver.

Treatment. Elimination of toxins should be promoted by securing a free action of the skin and bowels. The most hopeful line of treatment is to give intravenous saline containing 5 per cent. glucose by the continuous drip method described on p. 100, the treatment being continued for several days.

Subacute Necrosis of the Liver

This condition also begins in the same way as does catarrhal jaundice, but the jaundice persists for several months, and the liver, while remaining enlarged, gradually becomes noticeably harder. The spleen also becomes enlarged and firm. Whether bile pigments continue to appear in the urine depends on the degree of jaundice, but the stools soon regain their colour. The whole course is likely to be spread over several years ; jaundice of a mild degree may be constantly present, but more usually recurs at intervals, lasting for perhaps a few days or for several weeks, and disappearing completely between times. Clubbing of the fingers, polycythæmia, and cyanosis may slowly develop. Portal obstruction and ascites may appear terminally, and hæmatemesis may be a late complication.

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[1] Cholæmia is a toxic condition met with in conditions which involve considerable loss of liver function, and is analogous to uræmia in renal disease, but just as uræmia is not due merely to an accumulation of urea in the blood, so cholæmia is not due simply to the presence of bile in the blood. indeed fatal cholæmia may take place in the absence of jaundice.

At post-mortem examination the liver is enlarged and bile-stained. Its surface is nodular, the nodules consisting of clumps of regenerating liver cells (nodular hyperplasia). Between the nodes are troughs of atrophied liver tissue undergoing fibroblastic change. Among the fibrous tissue may be seen many new-formed bile canaliculi.

The outlook is poor. Death may come about during an acute recurrence of jaundice. Several instances have, however, been recorded in which survival has been maintained, probably owing to the formation of new and functioning liver tissue. Treatment is largely symptomatic. Long rest is essential, and a course of biliary antiseptics such as hexamine and salicylates should be given.

Cirrhosis of the Liver

Cirrhosis of the liver is rare in childhood. Forbes was only able to find 40 instances in a series of 5,500 post-mortem examinations on children at Great Ormond Street. The sexes are equally affected. The types of cirrhosis are similar to those of adults, and may be divided into : (i.) multilobular, or portal ; (ii.) unilobular, or biliary ; (iii.) pericellular ; the last being due to syphilis. While in adults the multilobular variety is the most common, in childhood the majority are of the biliary type. In 48 cases in children under twelve years of age, Poynton and Wyllie found biliary cirrhosis in 24, and syphilitic cirrhosis in 19.

Although the symptoms of cirrhosis in childhood show no great difference from those encountered in adults, different causes operate in the young. Alcoholism scarcely needs to be considered, nor are the common causes of obstructive biliary cirrhosis in adults, such as impacted gall stones or new growths of the head of the pancreas, met with in children.

Multilobular Cirrhosis. This, the rarest of the three main divisions of cirrhosis, may be present at birth. The youngest case observed by the writer was in a boy aged five months. The condition may affect successive members of a family. The origin of the disease is quite obscure, but it seems likely that maternal toxins elaborated during pregnancy may reach the fœtal liver and initiate the process.

The child may be brought on account of enlargement of the abdomen or because of failure to grow. On examination the liver is found to be considerably enlarged, and is firm and not tender, and the spleen is also enlarged. Telangiectases appear

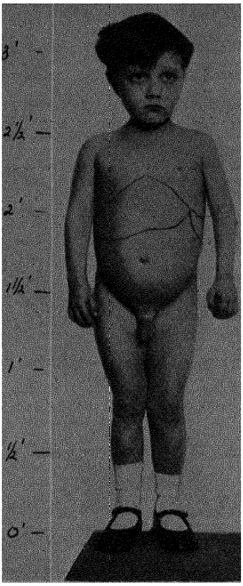

FIG. 40. Boy aged four years suffering from multilobular cirrhosis of the liver. The outlines of the liver and spleen are indicated.

on the cheeks and nose, the superficial abdominal veins may be distended, and ascites is likely to occur. If jaundice appears it only does so terminally.

The interference with growth becomes a more and more noticeable feature as the children get older, but although they become considerably stunted in height, they generally remain plump until towards the end of the illness. The mentality is unimpaired, although the physical handicap may lead to neglect of proper education.

The outlook is ultimately bad. Death from cholæmia usually comes about within ten years.

The treatment is purely symptomatic.

Biliary Cirrhosis. The two chief forms of biliary cirrhosis, one in association with congenital obliteration of the bile ducts, and the other accompanying subacute necrosis of the liver, have already been dealt with.

Pericellular (Syphilitic) Cirrhosis. This condition is considered in a later chapter ; suffice it here to say that it is one of the most common forms of cirrhosis to be met with in children. The condition may be present at birth, or may appear within a few weeks, and is likely to be accompanied by jaundice. Microscopical examination of the liver at this stage shows a fine network of fibrous tissue compressing and destroying the hepatic cells. Special staining methods will reveal the presence of numerous spirochætes.

Syphilitic cirrhosis may also appear in a tertiary form in older children. At this stage the surface of the liver is likely to be irregularly bossed and scarred from the presence of multiple gummata of varying sizes. Jaundice in these cases is variable, and is often a late symptom.

Fatty Liver

Fatty degeneration of the liver is a common finding at the post-mortem examination of children. It is almost always present, and to a severe degree, after death from acute gastro-intestinal disorders in infancy, and may in fact be the only abnormal feature of the autopsy. It is also often present in young children who die from acute infections such as bronchopneumonia, and is invariably present in diabetic coma. It is also found in the grave anæmias, and occurs in chronic venous engorgement of the liver.

It may be seen from the list given above that fatty degeneration of the liver occurs as a secondary condition, and is seldom likely to be diagnosed during life. That it plays an important part in determining the outlook of any illness in which it occurs cannot be doubted, and in the face of acute disease an attempt should be made to prevent it by seeing that the child has a plentiful supply of simple fluid, and such easily assimilable carbohydrate as glucose.

Von Gierke's Disease (Hepatomegalia Glycogenica)

This rare condition was first described by Von Gierke in 1929, and is due to an inability of the liver to rid itself of glycogen. The disease dates from birth. The child is likely to be brought on account of swelling of the abdomen, which on examination is found to be caused by a very enlarged and firm liver. The great size of the organ has been shown to be due to distension of its cells with glycogen. The spleen is not enlarged, which may help to distinguish these cases from cirrhosis of the liver, nor is jaundice a symptom.

Owing to the inability of the liver to give up its glycogen, the blood sugar is low, and as a result the oxidation of fats is incompletely carried out, and so traces of ketone bodies are constantly present in the urine. An injection of adrenalin fails to produce the customary rise of blood sugar. A certain degree of stunting gradually appears, which Ellis has suggested may be due to the constantly low blood sugar.

Most of the cases have died in the early years of childhood from some intercurrent infection.

Tuberculosis of the Liver

This is generally seen in its miliary form, the liver being affected in company with other organs as part of a generalised miliary tuberculosis.

As a rare condition large areas of caseating tuberculosis may develop in the liver (massive tuberculosis). It is generally thought that in these cases the bacillus reaches the liver by way of the bile ducts, giving rise to a tuberculous pericholangitis with the development of caseous areas in relation to the intra-hepatic bile passages. It is also possible that infection may reach the liver directly from the peritoneum, as seemed probable in an instance recorded by the writer. The caseous areas vary considerably in size, and may sometimes occupy nearly the whole of a lobe. Not infrequently they rupture into the bile capillaries, leaving a cavity which becomes deeply bile stained.

Massive tuberculosis of the liver usually lasts several months, and may give rise to no specific symptoms beyond considerable enlargement and irregularity of the liver. Jaundice is uncommon, and the condition is very likely to be mistaken for cirrhosis, but the presence of a chronic focus of tuberculosis elsewhere in the body, such as in the bones or joints, should arouse suspicion.

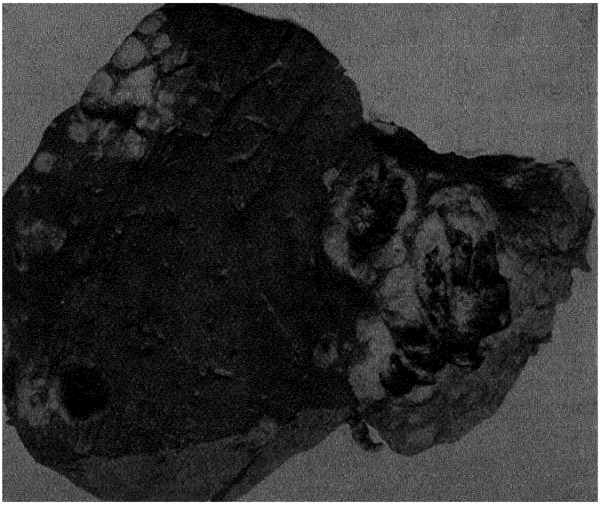

FIG. 41. Massive tuberculosis of the liver. The left lobe is almost entirely occupied by a large abscess cavity. Several caseous areas can be seen on the periphery of the right lobe. From a boy aged ten years.

The prognosis is bad, for the disease leads to a fatal issue after a year or so.

New Growths of the Liver

Malignant disease of the liver is very rare in children. Both primary sarcoma and carcinoma have been recorded, generally in the first year or two of life, and give rise to great enlargement of the organ. The condition is always fatal. Pepper's tumour of the suprarenal, which occurs during the first few months of life, also gives rise to malignant invasion of the liver. Innocent tumours such as angiomata and adenomata are occasionally met with at autopsy, but have no clinical significance. Congenital cystic disease of the liver is very rare, but has been recorded in association with congenital cystic disease of the kidneys. The cysts are of small size and do not give rise to symptoms during life.

DISEASES OF THE GALL BLADDER

Diseases of the gall bladder in childhood are rare. The great majority of the recorded cases are associated with cholelithiasis—this was so in 140 out of 226 instances of gall bladder disease occurring in children under fifteen years, collected by Potter,[1] the remainder being for the most part examples of acute cholecystitis. Cholecystitis as a complication of typhoid fever is also rare, but Rolleston and McNee refer to four children between three and ten years who were affected in this way.

Cholelithiasis. Gall stone formation in children is so rare as to be of little clinical importance. The majority of cases are found within the first few months of life, the stones being very small, black in colour, and consisting chiefly of inspissated bile. No treatment is called for. In the few recorded cases of gall stones in older children the symptoms have not differed from those in adults. They may occur in association with acholuric jaundice.

Acute Cholecystitis. This may arise from some blood-borne infection, or may be secondary to typhoid fever or appendicitis. The symptoms consist of pain and tenderness over the gall bladder, fever, vomiting, chills, and rigidity of the right upper quadrant of the abdomen. Jaundice is not usual. Treatment consists of warm applications locally to relieve the pain, salines (magnesium sulphate) by mouth, and such biliary antiseptics as salicylates or hexamine.

Chronic Cholecystitis. This may follow typhoid fever or may be the result of a blood-borne infection. In the case of a boy aged ten years[2] the symptoms consisted of recurrent attacks of severe abdominal colic with tenderness and rigidity over the gall bladder, which was enlarged and palpable. A faint icteric tinge followed the crises of pain.

Cholecystectomy offers the most satisfactory line of treatment.

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[1] Potter, A. H., *Surg., Gyn. and Obst.*, 1928, **46**, 705.

[2] Sheldon, W., and Edwards, H. C., *Lancet*, 1934, *i.*, 82.

DISEASES OF THE PANCREAS

Acute Pancreatitis. Except as a complication of mumps, acute pancreatitis is of great rarity in childhood. The condition is also uncommon after mumps, although during small epidemics its incidence has on occasion risen as high as 20 per cent. As a rule acute pancreatitis following mumps begins about a week after the onset of the parotitis, but in 5 out of 119 instances collected by Farnum the pancreatitis actually preceded the parotitis.

The symptoms consist of severe epigastric pain and tenderness, urgent vomiting, fever, and rigidity of the upper part of the abdomen, followed by signs of collapse. The outlook for cases that follow mumps is good; the symptoms begin to subside after a few days, and a recovery is usually made without the necessity of surgical intervention.

Treatment consists of complete rest, a light fluid diet, small doses of opium to relieve the pain and check the vomiting, and the application of an ice bag to the epigastrium.

Chronic Pancreatitis. This may arise in childhood as the result of chronic infections, and is most commonly met with in association with congenital syphilis, when small gummata may be present as well as a more diffuse interstitial fibrosis. The internal secretion of the pancreas is not as a rule affected. If clinical signs arise they will be due to interference with the external secretion of the gland, and may include steatorrhœa and the passage in the stools of undigested muscle fibres.

Cystic Fibrosis of the Pancreas. This is a congenital condition, in which the pancreas appears to be immaturely developed, the acini are reduced in number and some are distended into cysts, and there is a considerable increase of fibrous tissue. The islets of Langerhans are usually unaffected.

From an early age, and despite a good appetite, affected infants fail to thrive, and pass large loose offensive motions; on a normal intake of fat, liquid oil may separate from the stool (congenital steatorrhœa), and on analysis of the stool much of the fat is undigested. Although the symptoms bear a superficial resemblance to those of cœliac disease, the onset from birth, the presence of liquid oil in the stools, and the high percentage of undigested fat in the motions, are distinguishing features. The outlook is poor; death, usually from intercurrent respiratory infection, is likely to occur within two years.

CHAPTER XIII

DISEASES OF THE URINARY SYSTEM

SPEAKING broadly, the diseases of the urinary tract in childhood differ but little from those that affect adults, although in early life the symptoms are likely to be less easily localised, and therefore the routine examination of the urine becomes a most important part of the examination of a child. In infancy the amount of urine passed each day is relatively greater in proportion to the body-weight than in later years, largely because of the entirely fluid nature of the diet; at the same time, measurements from day to day show wide differences, probably because the amount of fluid lost by the stools is so variable.

The collection of a specimen of urine from young children sometimes presents practical difficulties. Infants are most likely to pass urine immediately on waking up or at the end of a feed, and therefore a specimen is most easily obtained at these times. In infancy also the bladder is more an abdominal than a pelvic organ, and because of this, slight pressure over the hypogastrium may start a flow of urine. If the specimen is required urgently a skilled nurse may employ other methods, for instance, in male infants a short stout test-tube can be attached to the penis by strapping, the test-tube resting between the thighs, and if the end of the penis is first thoroughly cleansed a specimen obtained in this way is sufficiently sterile for ordinary cultural purposes. In female infants a sterile specimen is easily obtained by passing a small rubber catheter. If a microscopical examination of the urine for pus cells is all that is required, as for instance in the diagnosis of acute pyelitis, the sterility of the specimen is not important, and it may then be sufficient to place the child on a mackintosh sheet spread over pillows in such a way that the centre of the mackintosh forms a pouch into which the urine can collect.

Pain arising from the urinary tract is not uncommon in infancy, but we may sometimes be misled by a story that the baby screams every time he passes water, for it may really be that he passes water every time he screams. It is quite likely that the rigid contraction of the abdominal wall when an infant is screaming is sufficient to compress the bladder and so lead to urination, just as an infant will often micturate when the hypogastrium is being palpated. In male infants a common cause of screaming during micturition is the presence of a small ulcer at the urinary meatus. It usually occurs in circumcised infants, and is partly the outcome of the unprotected glans chafing against a rough napkin. Other cases are associated with a very small urinary meatus of pin-hole size, and the ulceration is then likely to recur again and again. The ulcer is often so minute that close examination is required to discover it. The exudate from it dries into a scab over the meatus, and the bursting open of this seal makes micturition painful, and not infrequently a few drops of blood precede the passage of urine. Treatment consists of covering the glans with a piece of gauze freely spread with vaseline, and in paying careful attention to changing the napkins as often as necessary to keep the parts dry. It is also a good plan to rinse the napkins in a solution of boracic acid before they are dried. A pin-hole meatus should be enlarged by making a small downward slit in it, stitching back the sides to prevent the incision from quickly closing again.

Painful micturition may also be due to a highly acid urine coming in contact with reddened and excoriated buttocks. The diet will then require overhauling, especially cutting down any excess of fat or sugar, and small doses of potassium citrate should be given. The passage of crystals into the urine may also lead to considerable dysuria. The commonest offenders are uric acid crystals, which are excreted abundantly during the first few weeks of life, and may give rise to attacks of renal colic with screaming and vomiting. More rarely the passage of oxalate crystals accounts for the symptoms. Relief is given by increasing the intake of fluid and by giving alkalis.

ALBUMINURIA

A trace of albumin in the urine is found in many conditions, without amounting to nephritis. It is often present when there is high fever, and may thus be found during tonsillitis, pneumonia, and so on, and it is also present during acute or chronic cardiac failure.

Orthostatic Albuminuria (Postural, Cyclic, or Functional Albuminuria)

The essential feature of this condition consists of the passage of albumin in the urine after the child has been up for a few hours, although the specimen passed immediately on rising in the morning is free of albumin. As a rule the albumin is only present in traces, but it may be enough to form a thick cloud. Analysis of the protein shows that it consists largely of globulin.

Orthostatic albuminuria is most commonly met with from about the sixth year until puberty, and there is often a history that the child has been growing quickly and is showing signs of physical fatigue. One may frequently suspect the presence of this type of albuminuria merely by noting the stance of the child, who instead of being erect stands in a sagging, dejected attitude, the chin drooping on the chest, the chest flattened, the scapulæ sticking out, the abdomen bulging forward, and the normal lumbar lordosis being much exaggerated. Frequently other features are present which serve to increase the debilitated state. Pressure of school work, chronic infections, especially in the throat, a disordered digestion, and constipation, are not infrequently present. Anæmia, palpitations, and fainting attacks are likely to occur.

Several explanations of functional albuminuria have been advanced. The usual reason that is given is that it is caused by venous engorgement of the kidney, which may be due either to a general visceroptosis affecting the kidneys as well as the other viscera or to stretching of the renal vessels owing to the lumbar lordosis. That the assumption of the erect posture is in some way a contributory factor cannot be doubted, although Samuels [1] has pointed out that if food is withheld when the child gets up the albuminuria does not appear. A deficiency of calcium ions has also been blamed, and certainly the albumin may cease to appear after giving calcium salts. Others have pointed out that the albuminuria of nephritis may show the same relation to posture at a time when the nephritis is recovering, and therefore have looked upon orthostatic albuminuria as the relic of some slight previous kidney disease.

Diagnosis. The diagnosis is generally an easy matter. Specimens of urine should be collected before the child rises in the morning, at midday, and in the evening, and be tested for albumin. If more extensive tests of renal function are carried out they will be found to be normal. The outlook as regards kidney function is quite good, and as a rule the condition passes off at or before puberty.

Treatment. From the point of view of treatment orthostatic albuminuria should be regarded merely as a symptom of debility, and treatment must therefore be directed towards improving the general health. In the first place any foci of infection must be sought out and dealt with. The diet should be a generous one, and there is no need to withhold protein from it. A regular amount of daily exercise, short of fatigue, is valuable, and special exercises to improve the posture should be carried out. Careful attention must be paid to the regular action of the bowels, and a general tonic such as Easton's syrup (10 to 15 drops ter die) should be described. A short course of Calcium Gluconate or Calcium Lactate (gr. 10 to 15 ter die) is useful.

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[1] Samuels, *Amer. Jour. Dis. Child.*, 1929, 37, 367.

Other Abnormal Constituents

The passage of uric acid crystals during the first few weeks after birth has already been mentioned. Post-mortem examination of infants of two or three weeks of age commonly reveals heavy deposits of uric acid crystals in the connecting tubules of the kidneys, where they may be seen with the naked eye as bright yellow streaks converging towards the apices of the pyramids.

In older children deposits of urates in the urine are not uncommon during times of fever and sometimes after strenuous exercise, and may be sufficient to make the urine cloudy and stain the chamber pink. The popular notion that the passage of urates denotes a rheumatic tendency is incorrect, and the homely remedy of withholding meat from the diet makes no difference and is unnecessary. The condition rapidly subsides under a liberal supply of fluid together with small doses of potassium citrate. Oxalate crystals are occasionally present in small numbers and are then symptomless, but they may sometimes appear in showers and may then give rise to hæmaturia and renal colic. This is specially likely during the season of the year when rhubarb, spinach, and strawberries are available.

A child is occasionally brought on account of a strange colour to the urine, and in this connection it may be pointed out that the dyes used in colouring sweets may be excreted in the urine, and the urine may also be tinted if the child sucks dyed materials. A not infrequent example of this is a pale pink urine with a greenish efflorescence on the top, which comes from eating pink sweets dyed with eosin. The colour is quite unlike the red or smoky tint due to the presence of blood. A deep red colour to the urine may be due to the presence of free hæmoglobin, which can be distinguished from hæmaturia inasmuch as the chemical tests for blood are positive, but there are no red cells to be seen on microscopical examination. Hæmoglobinuria occurs in paroxysms lasting for a few days at a time, and the attacks are often preceded by exposure to cold. In childhood the majority of the cases are due to congenital syphilis.

The significance of pus in the urine is dealt with later under pyelitis, but a few words may be said here of bacilluria. In this condition bacilli are excreted in the urine in such large numbers that when a specimen is held up to the light a shimmering cloudiness can be seen. Bacilluria may occur at any age in childhood, although it is not common. The usual organism is the *B. coli*, although typhoid organisms or *B. proteus* are occasionally present. It is probable that the organisms have originally been derived from the bowel, but whether their continued appearance in the urine indicates a focus of infection in the urinary tract, or whether they are being constantly transferred from an unhealthy bowel, is uncertain. As a rule the onset is quite insidious, although there is sometimes a history of acute urinary symptoms at the beginning. Concomitant evidence of renal damage, such as pus, casts, or blood in the urine, is seldom forthcoming, and although the excretion of bacilli may go on persistently or intermittently for months or even years, the general health of the child is often not appreciably lowered. In other cases the intermittent appearance of bacilli may be accompanied by low fever.

Treatment. Simple bacilluria can sometimes be cleared up by giving urinary antiseptics such as hexamine (gr. 4 to 8), or salol (gr. 3 to 5). Liberal fluids should also be prescribed. Autogenous vaccines prepared from a culture of the urine are sometimes effective and in *B. coli* cases treatment with sulphanilamide or mandelic acid is worth a trial (see p. 299).

NEPHRITIS

Although several attempts have been made to find a satisfactory classification of the various types of nephritis, so far none has been evolved which gives complete satisfaction to the clinician, the pathologist, and the biochemist.

The difficulty is increased by the very function of the kidneys, which is to filter off from the blood the waste products of metabolism and other noxious substances which may find their way into the circulation. Loss of kidney function is therefore bound to have repercussions on the rest of the body, and recent investigations have shown that some of the symptoms hitherto ascribed directly to renal damage, such for instance as œdema, are in actual fact dependent on chemico-physical alterations in the blood and body tissues. Bearing this in mind, it becomes necessary to regard nephritis not as an isolated affair of the kidneys, but to take a broader outlook and view the renal damage as part of a more widespread change.

Turning to the kidneys themselves, a classification which suggests that damage has been inflicted mainly on the glomeruli, or the tubules, or the interstitial tissue, is unsatisfactory, inasmuch as evidence is generally forthcoming that when one of these tissues is involved the others are affected as well. Recently much interest has attached to the pathological conception that the renal changes are in some cases degenerative rather than inflammatory, and to these cases the term " nephrosis " has been applied. The outstanding clinical feature of these particular cases is the presence of gross œdema over a long period, and without evidence of nitrogen-retention in the blood.

Our purpose will best be served by considering nephritis under the following headings :—

Acute nephritis.
Chronic œdematous nephritis.
Renal fibrosis.

Acute Nephritis

Acute nephritis may be met with at any age in childhood, but is least common in infancy. It affects both sexes equally. It is now generally accepted that this form of nephritis is the result of damage to the kidneys by toxins which are usually derived from foci of bacterial infection elsewhere in the body, and the most common organism to give rise to these toxins is the streptococcus, but pneumococcal, staphylococcal, and other bacterial lesions occasionally form the starting point. The importance of infections as a forerunner of nephritis is undoubted, and just as an acute infection may be followed by an acute nephritis, so may recrudescences of a more chronic nephritis be associated with exacerbations in some chronic focus of infection. In childhood the most common source of infection lies in the nasopharynx, and it is a common story that acute nephritis has developed at an interval of a few days or a week or two after a sore throat. Of the specific fevers, scarlet fever is the one most likely to be followed by acute nephritis, and this might almost be expected now that we know that scarlet fever is to be looked upon as a special variety of acute streptococcal sore throat. Acute otitis media, and alveolar abscesses in connection with carious teeth, are two other common foci of infection which may be followed by inflammation of the kidneys, and staphylococcal lesions of the skin may also be the source of origin. Although a careful examination will generally reveal some such focus of infection, there remain a number of cases for which no adequate cause can be found. Exposure to cold and chills are cited as provocative causes, but they probably act simply by making the child more susceptible to infection. Occasionally chemical poisons such as phosphorus, mercury, and turpentine, etc., may take the place of bacterial toxins.

Symptoms. With regard to symptoms, acute nephritis may be conveniently described under two headings :

Acute nephritis without œdema.

Acute nephritis with œdema.

The difference between these two types is one of degree. In the former the lesion in the kidney principally affects the glomeruli, while in the latter the changes are more widespread. All gradations are met with, however, from the mildest cases, which are characterised by temporary hæmaturia, to the most severe cases which are accompanied by much œdema and retention of nitrogen in the blood, and culminate in uræmia and death.

Acute nephritis without œdema. This is a very common form of nephritis in children. The onset is generally brisk, and the outstanding symptom consists of the passage of bright red urine containing much blood. Apart from the hæmaturia, symptoms are surprisingly few and the general health is but slightly disturbed. Headache and vomiting may occur initially, and for a day or two the temperature may be raised a few degrees, but is as often normal. There is no pain or tenderness over the kidneys. Œdema is absent, and the retinæ show no changes. Investigation of the renal function shows that it is scarcely affected. The blood urea may sometimes be raised to 50 or 70 mgm. per 100 c.c. for a few days, but soon returns to normal, and is often not raised at all, and the urea concentration test is also unaffected. Blood pressure readings seldom show any rise, and the heart sounds are not altered.

The output of urine is not diminished. The amount of blood varies, but is sufficient to give a bright red or smoky colour to the urine. The loss of albumin is small, and is often no more than might be accounted for by the amount of blood, and as a rule the albuminuria seldom lasts much longer than the hæmaturia. Microscopical examination often shows a few leucocytes in addition to the red cells, and granular and hyaline casts may be present as well as an occasional blood cast. The urine is sterile on culture, which distinguishes this common form of nephritis from the more rare acute embolic nephritis—which is met with most characteristically in subacute bacterial endocarditis and in which streptococci or other organisms may be cultured from the urine.

Acute nephritis with œdema. This is a more serious form of nephritis than that just described, but is fortunately less common. The onset as a rule is sudden and is marked by fever, vomiting, headache, pains in the back, and the passage of scanty blood-stained urine. In other cases the onset is more insidious, and puffiness of the eyelids or œdema of the extremities may then be the first symptom. The œdema varies considerably in amount from a just discernible puffiness of the face and eyelids to a widespread pitting œdema affecting the whole body, and noticeable particularly in the upper eyelids, over the lumbar region, in the scrotum, and in the feet, while in the most severe cases there may be transudates of fluid into the serous membranes, giving rise to signs of hydrothorax and ascites. Epistaxis is not uncommon, the tongue is heavily furred, and the breath may be offensive. The stools are sometimes loose, but constipation is more usual. Examination of the fundi may show turgidity of the vessels, and even papillœdema. The blood pressure is often slightly raised, and the heart action may be noticeably deliberate and the aortic second sound may be accentuated. Evidence of an impaired renal function is indicated by the raised blood urea, which is usually about 60 to 70 mgm. per 100 c.c., but may be much higher, by a lowered urea concentration test, and a reduced urea clearance test.

The urine is almost always reduced in amount and looks concentrated, and is usually red or smoky from the presence of blood. There is a heavy cloud of albumin, which is much more than can be accounted for by the blood, and microscopical examination not only confirms the presence of blood cells, but may show numerous granular, epithelial, or blood casts.

It is in this form of acute nephritis that uræmia may develop, and it is always a serious complication. Its onset is attended by an increase of the previous symptoms. The secretion of urine becomes much reduced and may even fail entirely, headache increases, vomiting may become intractable, the respirations become rapid and may show a hissing quality due to acidosis, the blood pressure steadily mounts, and there may be some swelling of the optic discs. An increasingly unpleasant state of the mouth often suggests the onset of uræmia ; the tongue becomes dry and brown, sordes and ulcers appear on the lips and inside the cheeks, the breath becomes very offensive, and an ascending septic infection of the parotid glands may occur. Muscular twitchings may make an appearance, and convulsions may follow, followed by coma or delirium, or occasionally by local paralyses affecting the eyes, the face, or the limbs. Although fatal cases of acute nephritis generally terminate in uræmia, the presence of this complication does not necessarily imply a fatal issue, for with energetic treatment complete recovery may come about. Of other complications, bronchopneumonia is the most serious. Symptoms of cardiac failure are not often met with, and then are usually terminal.

Pathology. Owing to the almost invariable recovery of acute nephritis without œdema, opportunities of examining the kidney do not often arise. The changes chiefly affect the glomeruli, which are swollen and hyperæmic, and at a later date some of them become atrophied and replaced by scar tissue. Fatal cases of acute nephritis are almost confined to the type with œdema, and at autopsy all the structures in the kidney are involved. The kidneys to the naked eye are swollen and congested, the cortex is hyperæmic, while the medulla is pale. There may be petechial hæmorrhages under the renal capsule. Microscopically the glomeruli are swollen and the glomerular space may contain leucocytes and free blood, and the glomerular tuft may be adherent to Bowman's capsule. The tubules show a varying degree of swelling and desquamation of their epithelium, and the interstitial tissue may be œdematous and contain cellular infiltrations.

Diagnosis. The diagnosis of acute nephritis seldom presents any difficulty because of the definite findings in the urine. It may, however, be pointed out that puffiness under the eyes, which is a common symptom in childhood, is more often due to chronic digestive disorders than to nephritis, in which it is more noticeable in the upper than in the lower eyelids. The diagnosis of acute nephritis should not be considered complete until a thorough search has been made for any focus of infection from which the condition may have originated.

Course and Prognosis. In the acute type without œdema the outlook, both immediate and remote, is good, and the majority of cases recover completely. As a rule under treatment the blood disappears from the urine in a week or so, and the albuminuria also clears up quickly, although it occasionally happens that a trace of albumin reappears when the child gets up, and in this way may simulate orthostatic albuminuria. Sometimes, even with strict treatment, blood continues to appear in the urine for weeks or even months without any very noticeable deterioration in the child's general health. This almost invariably means that there is some persistent focus of infection from which toxins are continually reaching the kidney, and the hæmaturia may only cease when perhaps infected tonsils or dental abscesses have been dealt with. It follows that the presence of persistent hæmaturia should not be regarded as a contra-indication to dealing radically with a septic focus, but should encourage it. As already implied, this form of nephritis is liable to repeated attacks, so that the child may have as many as half a dozen recurrences. A small number of these recurrent cases eventually settle into progressive renal fibrosis with a rising blood urea.

Acute nephritis accompanied by œdema may terminate in various ways. Although the majority recover satisfactorily from the acute attack, the disease may run a rapid and fatal course ; this was so in 6 out of 23 cases collected by Moncrieff and Wyllie.[1] In general terms, the milder the symptoms the better is the chance of complete recovery, but even severe cases with uræmia and anuria may do well. In cases that are likely to progress favourably blood disappears from the urine within a week or two, the blood urea and blood pressure rapidly drop to normal, and the amount of albumin quickly diminishes from a thick cloud to a faint trace. Whether the persistence of a faint trace of albumin in the urine is consistent with cure is very doubtful, for with any fresh infection a relapse of nephritis is likely, and will cause further damage to the kidneys. In other cases the œdema persists, anæmia becomes more and more apparent, and the picture passes insidiously into that of chronic œdematous nephritis, to which may be added evidence of progressive nitrogen-retention with a slowly rising blood urea and a raised blood pressure.

Treatment. Absolute rest in bed is the first essential, and must continue until the urine is free of blood and until albumin has either disappeared or remains as only a faint cloud. In the mildest cases it may be only necessary for the child to remain in bed for about three weeks, but in cases with œdema rest in bed must generally continue for at least three months—and that in cases that are doing well. The patient should be nursed between blankets and carefully protected from draughts and chills, but as soon as the œdema and hæmaturia have disappeared the child may be allowed to rest in a sunny corner of a balcony.

The diet to begin with should consist of simple fluids such as water, barley-water, and well-sweetened fruit drinks, but these should be allowed in plenty. The amount of milk should be strictly limited owing to its protein and salt content, and for the first few days it is better omitted entirely, and from then until the urine is free from blood the daily amount should not exceed half a pint. When the hæmaturia has cleared, farinaceous foods such as rice, cornflour, custards, and gruels and bread and butter may be added, but salt should be carefully excluded until all traces of œdema have disappeared. Eggs, fish, and vegetables (cooked without salt) should be allowed as soon as the albumin has reached a persistently low level, and meat may be reintroduced after the child has been allowed to get up.

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[1] Moncrieff, A., and Wyllie, W. G., *Lancet*, 1926, *i.*, 128.

Drugs play only a small part in the treatment. During the acute stage a loose action of the bowels is desirable, and to obtain this half a drachm of pulv. jalapæ co. or a teaspoonful of magnesium sulphate may be given daily. Sweating should be promoted, although the amount of toxin lost in this way is probably small. A hot pack or hot-air bath may be given once a day when there is much œdema, and if at the same time a warm drink is given a profuse sweating may be obtained. During the hot pack a watch must be kept for signs of collapse, and stimulants such as brandy or camphor should be at hand. Although diaphoretics are not as popular as hitherto, it has seemed to the author that pilocarpine nitrate (gr. $\frac{1}{16}$ to $\frac{1}{10}$ hypodermically) at the beginning of the bath increases diaphoresis. Diaphoretic mixtures are generally without effect, but there is an advantage in giving alkalies to thwart any acidosis, and for this purpose gr. 10 each of potassium citrate and sodium bicarbonate may be given four times a day, and may with advantage be combined in a glucose drink. In the severest cases, when the cardiac action becomes rapid and weak, tinct. digitalis (m. 5), or tinct. strophanthi (m. 2), may be given twice or three times daily.

When the acute stage is over anæmia usually requires treatment, and should be dealt with by giving an iron preparation such as ferri. et ammon. cit. grs. 5–10 ter die. The return to a normal life should be made gradually, and a change of air to the country is desirable.

Lastly, the greatest importance must be paid to the search for any septic focus, for this may require treatment in order to bring the renal symptoms to an end. Otherwise it should be dealt with during convalescence in the hope of preventing any relapse of nephritis.

Treatment of Acute Uræmia. The treatment of acute uræmia merits a special description, although the rationale is the same as that underlying the treatment of acute nephritis. Simple fluids such as water and glucose-water should be given freely by mouth, but if persistent vomiting prevents this the stomach should first of all be washed out with a solution of sodium bicarbonate, at a strength of a drachm to a pint. Fluid may also be given per rectum by drip enema, after the bowel has been washed out. Venesection is a most valuable treatment in the face of oncoming coma. The amount to be withdrawn will depend on the age of the child ; half to one ounce will be sufficient during the first two or three years, while at the other end of childhood five or six ounces may be taken off. Convulsions are best treated by the withdrawal of cerebro-spinal fluid, but care should be taken to see that the fluid escapes slowly. If in spite of a lumbar puncture convulsions continue, a light anæsthetic may be given. Hot packs should follow these more urgent remedies. Drugs in uræmia are generally of little value, but magnesium sulphate has been recommended for the relief of cerebral œdema and convulsions, and may be given as a 50 per cent. solution per rectum, or as a 1 per cent. solution intravenously, allowing 10 c.c. for each kilogramme of body-weight. Sedative drugs such as chloral hydrate are also of value when the child is restless, and may sometimes check the vomiting. When the child has emerged from the uræmic state the treatment reverts to that of acute nephritis.

Chronic Œdematous Nephritis (Chronic Parenchymatous or Hydræmic Nephritis) (Nephrosis)

The essential features of chronic œdematous nephritis consist of a severe degree of œdema which persists for several months or may even drag on for a year or more, and the passage of a highly albuminous urine, but without cardio-vascular involvement or nitrogen-retention, at any rate until the later stages.

This form of nephritis is less common than the acute variety, and although it may occur in young infants it is seldom met with before three or four years of age. Both sexes are equally affected. Some of the cases begin as acute nephritis with œdema, which, instead of clearing up, gradually changes into the clinical picture of chronic parenchymatous nephritis, while other instances arise more insidiously without a history of preceding renal disease. Examination may sometimes show a chronic infection in some other part of the body such as the throat or middle ear from which toxins are presumably absorbed and damage the kidney, but at times no adequate cause can be found.

Symptoms. Puffiness of the eyelids or œdema of the feet is usually the first symptom, together with headache, lassitude, and pallor, and occasionally vomiting and even suppression of urine. The amount of œdema varies. It is usually so widespread that the whole child appears swollen, the face is puffed up and colourless, the eyelids may be so swollen that the lids can hardly be separated, there is œdema of the trunk, especially in the lumbar region, and the limbs are so œdematous that the skin may be shiny. The swollen tissues pit easily. Frequently the œdema is so severe in the penis and scrotum as to make micturition difficult. The peritoneum may contain several pints of free fluid, and there may be fluid in the pleuræ (hydrothorax) and pericardium. The fluid obtained on tapping the subcutaneous spaces is clear, but that from the serous cavities is often opalescent. Naturally with so much retained fluid the weight rises considerably, and regular weighings afford one of the best means of gauging the fluctuations in the amount of fluid. Not only may the degree of œdema be much more striking in one child than another, but the amount of œdema in any one individual may show remarkable fluctuations, a progressive accumulation being followed by a spontaneous diuresis and consequent loss of fluid. The position of the œdema may also vary; for instance, a diminution of ascites may be compensated by increased œdema of the legs, and for this reason measurements ot the abdominal girth as an indication of the degree of fluid retention are much less reliable than are alterations in the weight.

As a rule the temperature is scarcely raised, but bursts of pyrexia may occur for a few days at a time, and may indicate a lighting up of infection in the naso-pharynx or elsewhere, and are likely to be accompanied by increased œdema and the temporary passage of small amounts of blood in the urine. The bowels tend to be constipated, although loose actions may accompany the periods of fever. Anæmia of a secondary type is invariably present, although it may be to some extent hidden by the œdema, which may also mask the wasting which is slowly going on, indeed a sudden loss of œdema after a spontaneous diuresis is likely to reveal a surprising degree of emaciation.

The urine is diminished in amount, and the specific gravity is raised. There is a heavy albuminuria, sometimes to such an extent that the urine becomes almost solid on boiling. Microscopically hyaline, granular, and fatty casts, and occasional leucocytes, may be present, and at times there may be a few red cells, but these are not usual. There is seldom enough blood in the urine for it to be visible to the naked eye. Tests for renal function are often normal, that is to say, the blood urea and urea concentration tests are unaffected. Blood analyses show that the blood proteins are diminished, sometimes to as much as half the normal. The loss is of serum albumin rather than of serum globulin, so that the normal ratio between these two proteins is disturbed. The blood cholesterol is raised, and may be two or three times the normal. The blood chloride may also be raised while the œdema is increasing, but otherwise is below normal owing to the retention of chlorides in the body tissues.

The cardio-vascular system and the blood pressure are unaffected and the optic fundi appear normal, although if the disease lasts long enough the blood urea may begin to rise and the blood pressure may mount, changes which indicate that the kidney is undergoing a gradual fibrosis.

Pathology. At post-mortem examination the kidneys are swollen and œdematous, and bulge through the capsule as soon as it is incised. The capsule is not adherent. The cut section of the kidney shows it to be pale, but the relative proportions between the cortex and medulla are unaltered. Microscopically the changes chiefly affect the cells of the tubules, which are distended with lipoid material. In those cases to which the term "nephrosis" has been applied the glomeruli are unaffected, and this is indicated clinically by the absence of blood in the urine and the lack of any evidence of nitrogen-retention. It is only in those cases which have lived for some time, and in which cardio-vascular and azotæmic changes have gradually developed, that fibrosis appears at autopsy.

Much investigation has been recently carried out as to the mechanism at work in the production of the œdema. It is now generally held that the steady loss of blood protein through the kidney so alters the colloidal osmotic tension of the blood that water and chlorides escape from the blood into the body tissues, where they give rise to œdema. On this basis there are two methods of attempting to rid the body of its œdema ; firstly by giving a high protein diet in the hope of raising the blood proteins, and secondly by withholding salt from the diet in order to lessen the water-retaining capacity of the tissues. This explanation of the œdema does not, however, cover all cases, for instances occasionally arise in which gross œdema occurs without albuminuria.

Course and Complications. The course is a long one, covering months or years, and on the whole the eventual outlook is bad. A few cases—those in which there is no blood in the urine and no evidence of cardio-vascular change or nitrogen-retention—recover completely, but much more commonly if the œdema disappears it is gradually replaced by those other changes which indicate increasing renal fibrosis. Many cases do not progress to this extent because death overtakes them while they are œdematous and water-logged. Uræmia during the stage of severe œdema is uncommon, although there may be both lethargy and headache from cerebral œdema. There is, however, a definitely increased susceptibility to infections, especially pneumococcal, and death is frequently due to such complications as broncho-pneumonia or pneumococcal peritonitis or pericarditis. Ulcerative colitis is another complication which generally indicates a fatal outcome.

Death during the œdematous stage usually occurs within two years of the onset, but those cases that pass on to fibrosis may struggle on for several years, and during this time œdema may make temporary reappearances. Mention has already been made of the rapid disappearance of œdema which may accompany a spontaneous diuresis. Although this may lead to cure, the prognosis should be guarded, for it often happens that the diuresis proves to be but a phase in the disease and the œdema subsequently reappears.

Treatment. Rest in bed is necessary so long as there is œdema. The child should be nursed between blankets, and should be shielded as far as possible from such common infections as colds and sore throats. Foci of infection should be diligently sought for and eradicated.

In a disease which runs so long a course the diet needs to be supporting. When the œdema is severe some restriction of fluid intake is desirable, but should not be carried to an irksome degree. Salt should be strictly omitted from the diet both in the cooking as well as any ordinary table salt, and in addition bread should be baked without salt, and milk should be restricted to three-quarters of a pint a day. A high protein intake has been recommended by Epstein in the hope of raising the blood protein, and for this purpose fish, lean meat, peas, beans, and eggs should be given, provided that it has first been determined that the blood urea is not raised. In those cases in which œdema is accompanied by evidence of progressive fibrosis and a raised blood urea, foods which contain protein must be given more sparingly. Some have advised a limitation of fats, but there is little evidence that they are actually harmful, and they are probably of some value in lessening the likelihood of infections.

The use of drugs is mainly concerned with relieving œdema. A loose action of the bowels is desirable and should be obtained by daily salines or compound jalap powder. Thyroid has been recommended in combination with a high protein diet, and relatively large doses such as gr. 1 twice daily of dried thyroid may be given. Various diuretics may also be used, and of drugs of this class urea is one of the most serviceable. It may be given in drachm doses three times a day for a week or so, by which time if it is going to be of benefit diuresis will have occurred. It should not be continued unless it produces the desired effect. Diuretin (theobromine and sodium salicylate) may be given over a trial period of a week or so, gr. 5 ter die being a suitable dose, or theocin sodium acetate (gr. 1 ter die), or euphyllin (gr. 1 bis die) may also be tried. Mercurial diuretics should only be used when other drugs have failed. One of the best is salyrgan, of which ½ c.c. may be injected intravenously and repeated once or twice at intervals of four days. Big doses of alkalies have also been recommended, although the writer has not been impressed with their use. The citrates and bicarbonates of potassium and sodium may be combined in equal proportions, and as much as gr. 200 of alkali may be given in a day. Acid salts such as ammonium chloride have also been recommended.

Attempts may be made to lessen the œdema by sweating, and for this purpose hot packs and hot-air baths may be used. Skilled nursing is required, as there is a risk of collapse during the baths, and chilling must be carefully avoided. They are of more service in acute than in chronic nephritis, and in the latter have been largely superseded by dietetic and drug therapy. Mention must also be made of Edebohl's operation, which consists of stripping the capsules off both kidneys. The operation is sometimes followed by a considerable flow of urine and loss of œdema, but in other cases is without effect. The results of decapsulation were observed in 23 cases by Campbell,[1] who came to the conclusion that although the operation was sometimes followed by the disappearance of œdema there was no evidence of any improvement in the kidney function as judged by albuminuria or blood analyses. When the collection of fluid in the peritoneum or pleuræ is great enough to embarrass breathing or cardiac action, the withdrawal of fluid by tapping should be carried out. The disturbance caused in this way is sometimes sufficient to initiate diuresis.

Finally the anæmia which so commonly accompanies this form of nephritis calls for treatment with iron. Iron and ammonium citrate, grs. 5–10 ter die, is a suitable preparation.

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[1] Campbell, G., *Arch. Dis. Child.*, 1930, 5, 283.

Renal Fibrosis (Chronic Interstitial Nephritis)

This is the least common form of nephritis in childhood. As a rule no underlying cause can be found. A few cases occur as a late stage of chronic œdematous nephritis (secondarily contracted kidney), and rarely there may be a history of acute nephritis having occurred several years previously. Other instances arise in conjunction with chronic infections of the urinary tract, and are often associated with hydronephrosis or pyonephrosis occurring secondarily to some congenital deformity of the urinary apparatus which has led to partial obstruction to the outflow of urine. Renal fibrosis may certainly commence during intra-uterine life, and in such cases the kidneys at autopsy may be so shrivelled and fibrotic as to cause wonder that they should ever have been able to support life. Congenital cystic disease of the kidneys is also associated with considerable fibrosis.

Symptoms. The symptoms are for the most part similar to those met with in adults. The onset is insidious, and the child may be brought for a variety of symptoms such as enuresis, headache, vomiting, failure of growth, or even the nervous manifestations of uræmia. When the complaint is of enuresis, further enquiry will show that there is also frequency of micturition associated with polyuria and an almost insatiable thirst. Headache is a common complaint, and attacks of vomiting occur from time to time, particularly in the early morning. Cardio-vascular changes occur in many cases, but by no means in all. They are seldom evident when the disease arises insidiously or before birth, and are most marked in examples of mixed nephritis—thus in a child of nine years who had had a history of nephritis for several years and had at times had much œdema, the systolic blood pressure a few weeks before death was 240 mm. Hg. When the blood pressure is raised to this extent the aortic second sound becomes loud (normally in childhood the pulmonary second sound is louder than the aortic), and at autopsy concentric hypertrophy of the left ventricle is present. Retinal exudates and hæmorrhages and even optic atrophy may occur, but as a rule the fundi show no changes.

The daily excretion of urine is much increased, and the urine is pale and has a low specific gravity of about 1,005. There is seldom more than a faint cloud of albumin, and at times albumin may be absent from several consecutive specimens. Granular and hyaline casts can generally be found, and an occasional red cell may be present.

Renal function tests show considerable impairment. The urea concentration test is low, down to about 1 to 1·5 per cent. (normal 2 to 4 per cent.). The urea clearance may drop as low as 10 per cent. (normal being at least 80 per cent.). The blood urea is raised, and may be as much as 200 to 400 mgm. per 100 c.c. The blood phosphorus may be three or four times the normal, while the blood calcium may be lowered.

The striking feature in most children with this condition is the stunting of growth, which gradually becomes more evident as the child gets older, and with it the child has a yellowish waxy complexion and a dry skin. Failure to grow affects both the height and weight, and is generally quite obvious by the third or fourth year. The diminutive stature is often accompanied by skeletal deformities which on clinical, radiographic, and histological appearances are indistinguishable from rickets, and under the term "renal rickets" they form one of the types of "late rickets." These deformities generally make their appearance a little later than the dwarfing, and may not appear until towards the end of the first decade. The most obvious deformity consists of progressive knock-knee, but enlargement of the epiphyses of the long bones and beading of the ribs also occur. Renal rickets, of which a fuller description is given on p. 140, is most commonly met with in the insidious type of interstitial nephritis dating from birth, although it has been recorded in chronic nephritis acquired soon after birth, and it is occasionally a complication of congenital cystic disease of the kidneys.

The most important complication is uræmia. The symptoms are usually those of *chronic* uræmia, and consist of increasingly severe headaches, vomiting, disturbances of vision, sordes and offensive breath, and fine tremors. Convulsions may occur at any time, and are always of serious import. In other cases an exacerbation of acute nephritis occurs and is likely to lead quickly to a fatal acute uræmia.

Pathology. The kidneys have a variable appearance. There is a type which dates from before birth, and which during life may run a long course without cardio-vascular changes, and at autopsy the kidneys are found much shrunken, but the capsule strips readily. Congenital hypoplasia of the kidneys aptly describes these. In the type which is acquired after birth the kidneys conform to those met with in adults ; they are shrunken and granular, the surface may be dotted with small cysts, the capsule is adherent, and the cortex is relatively more shrivelled than the medulla. Microscopically in both types there is a diffuse increase of fibrous tissue producing much distortion of the normal architecture.

Course and Prognosis. The outlook for the child with chronic renal fibrosis is thoroughly bad. The majority do not reach puberty, and a fatal termination invariably ensues before the end of the second decade. Death usually comes about from uræmia, and symptoms of chronic uræmia may make fitful appearances for several years before the final attack. Acute infections such as broncho-pneumonia or pericarditis occasionally account for death. Cerebral hæmorrhage is a very rare mode of termination.

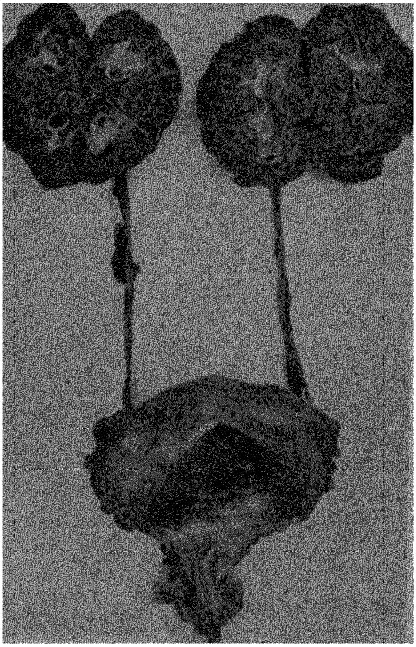

FIG. 42. Congenital cystic kidneys from a boy aged twelve years.

Treatment. Treatment is unsatisfactory inasmuch as there are no curative measures. Care should be taken to prevent infections, and the daily routine should be so regulated as to avoid fatigue, but prolonged rest in bed is not indicated, and these children are generally better when they are up and about. The diet needs to be simple and nutritious. Highly nitrogenous foods such as meats and fish should be omitted, but rigid dietetic restrictions are uncalled for. Fluid should be allowed freely to satisfy the constant thirst. When uræmia threatens, fluid with glucose should be given in plenty, and the withdrawal of blood and of cerebro-spinal fluid may temporarily postpone the ultimate issue. When renal rickets appears the ordinary methods of dealing with rickets are without effect. Genu valgum may become so severe as to interfere with the child's activities, and an attempt may be made to prevent this by the use of light orthopædic apparatus.

Congenital Cystic Disease of the Kidneys

This is an uncommon condition in childhood. It affects both kidneys, and is attributed to a developmental failure of the normal union between the secreting and the collecting tubules. The secreting tubules then dilate to form cysts. A diffuse fibrosis is always present, and is thought by some to date from intra-uterine life and possibly to account for the imperfect development of the kidneys. The disease shows hereditary and familial tendencies. In one type the cysts are relatively few in number, but attain to great size and may give rise to large abdominal tumours. These are more frequently met with in adults. In the type that gives rise to symptoms in childhood the cysts are usually smaller, but the kidney is riddled with them and resembles a firm sponge. Such kidneys may give rise to hard nodular tumours in each loin, but the symptoms for which the child is brought are usually those of renal fibrosis. Great thirst, polyuria, albuminuria, and dwarfing, are generally present. The ultimate prognosis is bad, and death may come about from uræmia in early infancy or towards the later years of childhood. It is only when the renal function is less interfered with that symptoms are delayed until adult life. Treatment can only be on the same lines as for renal fibrosis.

Urinary Calculi

Urinary calculi are uncommon in early childhood, but increase in frequency as age advances. It is somewhat surprising that calculi should be so rare in young babies seeing that during the first few weeks of life large numbers of uric acid crystals are passed in the urine. Post-mortem examination on infants who have died during the neonatal period often shows uric acid concretions no larger than a split pea in the renal pelvis, but during life these are probably dissolved or broken up into smaller fractions, for they seldom appear in the urine.

Calculi are more common in boys than in girls. For the most part they are composed of a mixture of urates and oxalates, and if they have lain in the bladder for any length of time they are likely to be encrusted with phosphates. Pure uric acid stones and cystin stones are rare varieties. Conditions which are likely to give rise to urinary stasis are important factors in stone formation, and calculi may therefore be found complicating the various congenital deformities of the urinary tract, most of which give rise to some degree of urinary obstruction. Stones may also form when children have to spend months on their back, as, for example, in the treatment of spinal caries. Chronic infection of the urinary tract is a common accompaniment of stone, indeed it is likely that such infection may contribute to calculus formation.

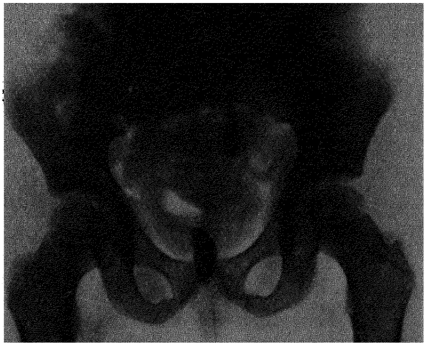

FIG. 43. X-ray of a boy aged six years, showing a calculus in the bladder, and another at the lower end of the left ureter.

Symptoms. The symptoms are similar to those met with in adults. Occasionally stones turn up unexpectedly at autopsy without having caused symptoms during life, at other times the symptoms may simply be those of chronic urinary infection associated with feverish attacks, frequency, painful micturition, and the passage of urine turbid with pus. Attacks of renal colic may occur, and are characterised by sudden intense abdominal pain radiating downwards from the loins, associated with restlessness and vomiting. There is considerable tenderness on the affected side, and the attack is likely to be followed by the appearance of blood in the urine. When the calculus is in the bladder micturition may be frequent and painful, or the flow of urine may be suddenly checked owing to the stone blocking the urethral orifice. The persistence of calculi in the urinary tract for any length of time is likely to give rise to hydronephrosis or pyonephrosis with disorganisation and destruction of kidney tissue.

Diagnosis. When calculi give rise to sudden symptoms such as colic or interrupted micturition they are hardly likely to be overlooked, but the possibility of their presence should also be remembered when there is persistent infection of the urinary tract. A combination of radiography, cystoscopy, and pyelography will enable the diagnosis to be confirmed.

Treatment. This is a surgical matter. During attacks of colic hot fomentations may be applied to the affected side, and a hypodermic injection of morphia (gr. $\frac{1}{12}$ at six years) may be required.

Tumours of the Kidney

The kidney is one of the most frequent sites of primary tumour formation in young children, and the majority of cases occur before the fifth year. The most common tumour is one composed of a mixture of tissues containing sarcomatous connective tissue, muscle fibres, and adenomatous elements. Such tumours result from the inclusion in the kidney of mixed embryonal elements, and they are almost always malignant. Extensive hæmorrhages into the growth may take place. In appearance the tumour is generally spherical, and grows at first within the renal capsule, which it distends, often stretching out the adrenal body into a thin layer. Removal of the tumour at this stage may lead to complete cure. Later on the kidney capsule becomes penetrated, and secondary deposits of growth then develop elsewhere, especially in the liver and lungs.

Symptoms. For some time the general health of the child may be but little affected, and the first complaint may be of abdominal distension or of hæmaturia. On examination the tumour is usually about as large as a cricket ball, but may be so big as to fill the loin and bulge the abdomen. It is generally firm, slightly mobile, and not tender. Wasting is a late symptom, being deferred until the formation of secondary deposits.

Diagnosis. The position of the growth may resemble a retroperitoneal sarcoma or a suprarenal neuroblastoma. As a rule in the former the course is a more rapid one, and the general effects on the child are soon evident, while in the latter the early appearance and characteristic situation of the secondary deposits in the skull or liver are diagnostic. There may be difficulty in distinguishing a new growth of the kidney from hydronephrosis, and this may only be decided at operation. The physical signs of the tumour should prevent it being mistaken for a mass of tuberculous glands or a chronic intussusception.

Treatment. The only effective treatment is surgical removal of the tumour, but if this is to be undertaken with any hope of permanent success it must be done before the capsule has become penetrated, and even so a fatal recurrence may take place.

URINARY INFECTIONS

Acute Pyelonephritis (Acute Pyelitis)

The term "acute pyelitis" implies acute infection of the urinary tract, generally by the *Bacillus coli*, and the illness is characterised by a sudden onset with high fever and the passage of pus and organisms in the urine. It may be doubted, however, whether the term "pyelitis," meaning an inflammation of the kidney pelvis, is strictly correct, and recently good reasons have been advanced to show that the initial lesion is within the kidney substance, and that the condition should be more properly regarded as an acute focal suppurative nephritis. The term "acute pyelonephritis" will be used here in preference to acute pyelitis. Symptoms of acute cystitis, such as frequency, pain on micturition, and tenderness over the bladder, are sometimes added to those of pyelonephritis, and the term "pyelocystitis" would then be a suitable one.

Etiology. *Age.* Acute pyelonephritis may occur within a few days of birth, and more than half the cases are met with in the first year. The incidence then falls rapidly and remains low during the later years of childhood.

Sex. There is no doubt that the condition occurs more often in female children, but it must not be supposed that the incidence is almost confined to girls. In the newborn there is actually a majority of cases in boys, and throughout the first year boys and girls are affected in equal numbers. The preponderance in girls applies to the period after infancy, and even then it is likely that the condition is more often diagnosed in girls because we have been taught to regard it as so much more common in that sex. If fever of unknown origin arises in a little girl, the urine is usually examined for pus, while under the same circumstances in a boy the possibility of pyelonephritis may be overlooked, and therefore the first essential in the diagnosis, namely, the microscopical examination of the urine, may be omitted.

Symptoms. The onset is usually sudden, and the child is quickly ill with a high temperature up to 103° F. or more. Vomiting commonly occurs at the beginning, and may continue throughout the course of the illness, and there may be one or more convulsions. Although not common, shivering attacks or rigors are sometimes an early symptom, and they are so infrequently met with in other diseases in children that their occurrence should always prompt an examination of the urine for pus. An upset of the bowels, either constipation or more frequently the passage of offensive undigested motions, is usually present, and enquiry will often show that the bowel disturbance has been present for a few days previously.

As the disease progresses the temperature remains high, often showing a daily swing of 3 or 4 degrees, and the child rapidly becomes pale, refuses food, loses weight, and is peevish and irritable. The irritability is sometimes a most prominent feature and one of which the parents may complain, and of itself should be sufficient to suggest the correct diagnosis ; occasionally it is accompanied by some stiffness of the neck and a positive Kernig's sign, which may lead to confusion with tuberculous meningitis. At times sharp attacks of pain occur in which the child screams out, stiffens, and appears anguished, and afterwards is left pallid and collapsed. Such bouts of pain suggest renal colic, and are probably due to the passage of clots of pus down the ureter.

The irritability of the child often makes physical examination difficult, and usually little is to be found beyond perhaps some slight tenderness over the loins, which is recognised by the child wincing when these parts are gently palpated.

In the newborn the onset is likely to be more indefinite, and restlessness, vomiting, diarrhœa, and diminished urinary output may be the chief symptoms. There may be periodic attacks of collapse in which the face appears grey and drawn, the lips are livid, and the infant feels cold. Such attacks indicate a severe infection, with a poor prognosis. Convulsions at this age are also a bad sign.

The diagnosis is made on the examination of the urine. It is seldom diminished in amount, but is almost always strongly acid. The most important constituent is pus, and the only satisfactory method of finding pus cells is to make a microscopical examination. The pus cells can easily be seen under a $\frac{1}{6}$th lens; they vary in number from 3 or 4 in a field up to a 100 or more, and may sometimes cause the urine to appear turbid, but it should be remembered that a urine which looks clear may none the less be found to contain pus. The number of pus cells often varies considerably from day to day, suggesting that the discharge of pus from the kidney is intermittent, and another curious feature is the way in which the pus cells often run together to form clumps of 50 to 100 cells. It must also be mentioned that pus cells are occasionally found in specimens from young children who are otherwise quite well, and this is particularly so in little girls. In girls there should be at least two pus cells per $\frac{1}{6}$th field of a non-catheter specimen of urine to be of any significance.

Blood in the urine is sometimes present in the early stages, but is seldom sufficient to colour the specimen, and casts are as a rule absent. Culture of a specimen of urine which has been collected with sterile precautions gives a growth of *B. coli* in almost all cases, but occasionally other organisms such as *B. proteus* or the pyogenic cocci may be present.

Diagnosis. Acute pyelonephritis may be overlooked because of the difficulty of collecting a specimen of urine from a young child, but with patience and the employment of such methods as have already been mentioned at the beginning of this chapter a sample can always be obtained. Unless it is proposed to make a bacterial culture, the sample need not be sterile, and in practice the finding of pus cells is of chief importance. The urine should be examined for pus in any infant who has a sudden feverish illness for which no adequate cause can be found.

Various conditions may be simulated by pyelitis. Confusion with tuberculous meningitis has already been mentioned, but although examination of the cerebro-spinal fluid will certainly help to exclude meningitis, it is simpler first of all to examine the urine, which is at the same time less disturbing to the child. In young infants the vomiting and looseness of the motions may seem to indicate that the whole trouble lies in the gastro-intestinal tract, and it not infrequently happens that the correct diagnosis is not made for some days until treatment directed towards the intestines has failed to bring relief. It must also be remembered that screaming and irritability are symptoms that commonly accompany acute inflammation of the middle ear in young children.

Pathology. At post-mortem examination the changes principally concern the kidneys. They are slightly swollen, and when the capsule is removed numerous small abscesses can be seen projecting on the surface. Section of the kidney shows these abscesses to be distributed chiefly in the cortex or between the cortex and the medulla, and there may be thin streaks of pus draining down towards the apices of the pyramids. Microscopical examination shows that the abscesses are situated in the interstitial tissue. Changes in the renal pelvis are difficult to make out, and seldom amount to more than some slight congestion and swelling of the mucosa.

The post-mortem findings certainly suggest that the condition should be regarded as one of multiple, focal, suppurative, interstitial nephritis (Chown ; Wilson and Schloss).[1] The absence of clinical evidence of disturbed renal function, such as œdema, suppression of urine, or the passage of casts, is attributed to the fact that the inflammation is interstitial and leaves the glomeruli and tubules unaffected, the presence of pus in the urine being accounted for by the bursting of the abscesses into the tubules and the discharge of their contents in this way. When recovery takes place, healing is complete, for in the few cases that have died for some other reason after acute pyelonephritis the kidneys have appeared healthy. An alternative view is that the inflammation primarily affects the renal pelvis, and that a fatal issue only comes about when the infection spreads upwards into the kidney substance to produce the changes which are found at autopsy, but even in fatal cases it is often impossible to demonstrate any change in the renal pelvis, nor does the position of the abscesses in the kidney support this view.

Much discussion has centred round the route by which infection reaches the kidney. Three routes have been suggested :—

(1) An ascending route *viâ* the bladder and ureters or periureteral lymphatics.

(2) From the bowel *viâ* the blood stream.

(3) From the bowel by direct extension to the kidneys *viâ* the lymphatics. There is no good evidence in favour of this view.

The high incidence in female children is a point in favour of the ascending route, although the infrequency of symptoms of cystitis is to be borne in mind. The fact that the majority of cases occur in infancy has been ascribed to the ease of ascending infection from the contact of soiled napkins against the vulva, but this argument would only be valid if the sex preponderance in females was particularly noticeable in infancy. It has, however, already been pointed out that the sex incidence at this age is practically equal, a fact which militates against the theory of an ascending infection. The high incidence in infancy is more probably due to the fact that this is the age when gastro-enteritis is most common. In favour of the hæmatogenous route the sudden onset with high fever and rigors may fairly be advanced, while the post-mortem appearance of the kidneys also favours this view. Not improbably infection takes place by both these routes, blood stream infection affecting boys and girls in equal numbers, while the extra number of cases in girls may be due to ascending infection.

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[1] Chown, B., *Arch. Dis. Child.*, 1927, **2**, 97 ; Wilson, J. R., and Schloss, O. M., *Amer. Jour. Dis. Child.*, 1929, **38**, 227.

Course and Prognosis. Acute pyelonephritis is a progressive condition, and the longer treatment is delayed the worse is the prognosis. With treatment the mortality is low. In 117 instances collected by Neale the mortality was just under 7 per cent. The presence of any congenital anomaly in the urinary tract not only increases the chance of a fatal issue, but also makes it more likely that the infection may continue in spite of treatment. Chronic pyuria persists in about 12 per cent. of the cases. When recovery takes place the symptoms generally subside within a few days of treatment, and as a rule the child is up and about in three or four weeks. For these children the ultimate outlook is good, and the illness does not give rise to permanent impairment of the renal function. In a different class are the children who have frequent relapses at intervals of several months. In the majority of these the infection has never entirely cleared up, and further investigation of them will often bring to light some congenital deformity of the urinary apparatus which has led to urinary stasis.

Treatment. The child must be nursed in bed. So long as there is fever the diet needs to be a light one, and such fluids as water, glucose-water, and barley-water should be given in plenty, for a copious flow of urine must be aimed at. If fluid is not tolerated by mouth it should be given per rectum or subcutaneously. Fruit drinks, stewed fruit, and milk puddings may also be given if they do not provoke vomiting. Careful attention should be paid to the bowels, constipation being relieved either by a mercurial purge or by enemata. Often the stools are loose and unhealthy, and then after an initial purge a mixture of salol and castor oil, such as the one on p. 208, will be useful.

In the usual type of pyelitis due to *B. coli* infection with a strongly acid urine, the most successful line of treatment is to make the urine alkaline as soon as possible with potassium citrate. To begin with the dose should be large and should be frequently repeated; for instance, to an infant gr. 10 may be given every two hours in a simple mixture, and for an older child gr. 30 two-hourly would be suitable. The following prescription serves very well:—

℞. Potassii citratis gr. x–xxx.
Sodii bicarbonatis gr. v.
Syrupi simplicis m. xv.
Aquam chloroformi ad ʒi.

The nurse should be instructed to test with litmus paper each specimen of urine as soon as it is passed, and the dose of alkali should not be lowered until the urine has been definitely alkaline for seventy-two hours. Unless alkali is pushed in big doses the reaction of the urine may hover round neutral for several days without any lessening of the symptoms. The effect of making the urine alkaline is often dramatic, for about the time that the reaction of the urine changes the temperature falls rapidly and the child's irritability and misery quickly clear up. When the urine has been alkaline for three days the dose of potassium citrate should be given every four hours, and this should be continued for a week, and then may be cut down to three times a day, provided that this is sufficient to keep the urine alkaline, for it is desirable to maintain the alkaline reaction for at least three weeks from the beginning of treatment. As a rule potassium citrate is tolerated very well, but should it provoke diarrhœa it may be replaced by sodium and potassium bicarbonate.

The amount of pus in the urine generally diminishes rapidly when the temperature settles, but a small amount may continue to be passed for a fortnight or so. It occasionally happens that the symptoms are only partially relieved by alkaline treatment, and then if it is persisted with the urine may become offensive. A change should then be made to sulphanilamide therapy, which is more fully considered on p. 299. There is no doubt that sulphanilamide is usually an effective remedy, and the question therefore arises whether it should be used in preference to alkaline treatment. In alkalies, however, we also have an effective remedy for the majority of cases, and a remedy which is at the same time harmless, whereas sulphanilamide is known to be capable of exerting toxic effects. It is therefore the author's practice to employ alkalies in large doses, and to reserve the sulphanilamide group of drugs for those cases that prove resistant to alkaline therapy, and for children with chronic urinary infection.

Chronic Pyuria, and Congenital Deformities of the Urinary Tract

The important part which congenital deformities of the urinary tract play in maintaining urinary infection has recently received considerable attention, and it is now generally recognised that the term " chronic pyelitis " as applied to a continual loss of pus in the urine is inaccurate and misleading. The term " chronic pyuria," although it only indicates a symptom, is perhaps preferable, for it implies the need of fuller investigation to determine the cause of the condition.

Deformities of the urinary tract may give evidence of their presence in various ways. They may, for instance, give rise to chronic distension of the bladder and an overflow incontinence, as, for example, when the outflow of urine is obstructed by mucosal valves in the urethra or by neuromuscular disorders at the neck of the bladder. In other cases the deformity of a ureter may cause retention of urine in the renal pelvis and may thus give rise to a palpable hydronephrosis, while if the deformity is bilateral, or is so placed as to cause partial obstruction of both ureters, the effect will be to produce double hydronephrosis with progressive renal fibrosis, and then the various symptoms associated with chronic nephritis and a failing kidney function will be present. Finally, all deformities of the urinary tract in children make the chance of *B. coli* infection more likely, and, when once infection has occurred, it is always most difficult to eradicate it. Not uncommonly the presence of a deformity is first suspected when, after a child has been diagnosed as suffering from acute pyelonephritis, the ordinary treatment fails to bring cure, and, although the symptoms may be to some extent relieved, the discharge of pus in the urine continues.

Post-mortem examinations of children show that congenital urinary abnormalities occur in about 2 per cent. of autopsies (Hyman). In 400 post mortems performed by the writer in children under twelve years of age, urinary deformities were present in 2·3 per cent. Roughly half of the cases show hydronephrosis affecting one or both kidneys, and depending on the site of the obstruction one or both ureters may also be tortuous and dilated, sometimes to such an extent that at post-mortem examination they resemble coils of small intestine.

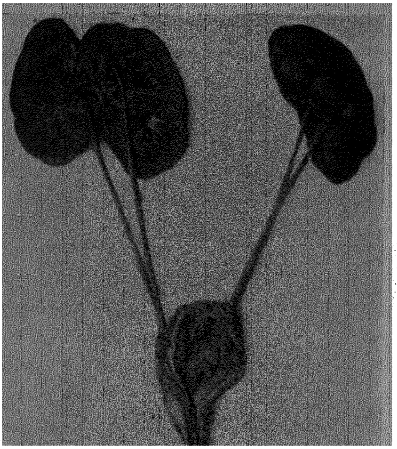

FIG. 44. Bilateral double ureter. From a child five months old.

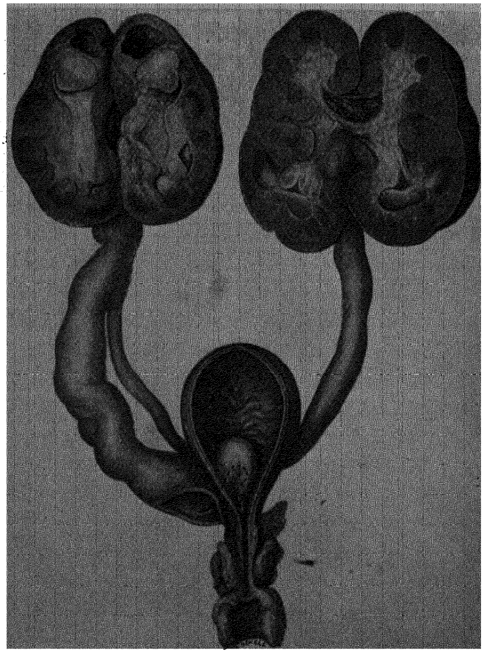

FIG. 45. Duplication of the right ureter. The extra ureter is dilated and drains the hydronephrotic upper pole of the kidney.

Types of Deformity : Kidney. Congenital absence of a kidney occasionally occurs, and then the opposite kidney hypertrophies and performs the work normally done by both kidneys, and symptoms seldom arise. In other cases the kidney is represented by a rudimentary fibro-fatty mass, and the ureter may merely consist of an impervious fibrous cord. Fusion of both kidneys to form one horse-shoe kidney sometimes occurs ; in such cases there are generally two distinct pelves and two ureters, but their anatomical relations are such that some degree of urinary obstruction usually arises.

Ureters. The most common abnormality of the ureters is a variation in their number. The ureter and kidney pelvis may be duplicated on one or both sides, each ureter draining its own portion of the kidney. The ureters may fuse before they reach the bladder or they may open by separate orifices, in which case one of the ureteric openings will be abnormally situated—it may even open into the bulbous urethra—and not infrequently the abnormally situated ureter is partially obstructed so that it becomes distended and the half kidney which it drains becomes converted into a hydronephrosis. Stricture of a ureter is another deformity, which may be either unilateral or bilateral. The site of the narrowing may be at the junction of the ureter and renal pelvis or at the lower end just as the ureter passes into the bladder. Stricture in the former situation may be associated with an aberrant branch of the renal artery which passes across the uretero-pelvic junction to supply the lower pole of the kidney. Ureterocele, that is, a prolapse of the ureter into the bladder, may also occur.

Bladder. Ectopia vesicæ is a rare deformity in which the anterior wall of the bladder and adjacent abdominal wall is unformed, and the posterior wall of the bladder is exposed to view. The pubic bones remain ununited. Ascending infection is a constant menace and accounts for a high mortality. Congenital diverticulum of the bladder is another rare deformity ; the stagnant urine in the diverticulum is likely to become infected, and the symptoms are those of chronic cystitis. The condition of congenital hypertrophy of the bladder has special clinical features of its own and therefore a separate description of it is given at the end of this section.

Urethra. The male urethra is a much more complicated structure than that of the female, and is much more subject to errors of development. Occasionally the orifice of the female urethra is misplaced, but other deformities are rarely met with.

As to the male urethra, epispadias and hypospadias may occur —conditions in which the urethra opens respectively on to the dorsum and undersurface of the penis. These conditions are purely surgical matters and call for plastic operation. Valve-like formations of the urethral mucosa sometimes occur, and are the most common cause of congenital hypertrophy of the bladder. Stenosis of the external urinary meatus is not at all uncommon, and may cause much straining when urine is passed. The condition is obvious on examination, and is relieved very simply by incising the meatus.

General Symptoms of Urinary Deformities

The symptoms produced by urinary deformities will naturally vary according to the nature of the abnormality. Such conditions as rudimentary kidney, horse-shoe kidney, and double ureter may not cause symptoms at all. On the other hand, some deformities may give rise to so much distortion and destruction of the kidneys during intra-uterine life that the child only survives for a few days. This was so in a child who died when three weeks old from uræmia, and at post-mortem examination bilateral hydronephrosis was found with gross destruction of kidney tissue brought about by a valvular obstruction in the urethra.[1] The changes were so advanced that they could not possibly have been produced merely since birth, and they incidentally afforded very good evidence, if such were needed, that secretion of urine actively goes on during fœtal life.

In many cases symptoms do not arise until infection has taken place, and this may be delayed for months or years after birth. The symptoms will then at first be those of acute pyelonephritis, but it often happens that the initial acute onset of infection is overlooked and then the child when first seen will be found to be suffering from pyuria of indefinite duration. When this is the case the child is likely to be thin and pale, and there may be a history of low fever which has been interrupted every few weeks by exacerbations up to 103° F. or so, together with vomiting and possibly one or more convulsions. During these bouts the child rapidly loses what little ground has been made since the last attack, and the urine may become noticeably cloudy with pus, and offensive. The amount of pus varies considerably, often there is enough to give a thick deposit if the urine is allowed to stand, while at other times the urine may appear clear to the naked eye although the microscope shows the presence of pus cells. Albuminuria is also present to a varying degree. The reaction of the urine is usually acid, especially if the infecting organism is the *B. coli,* but it may be alkaline when there is chronic cystitis.

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[1] King, H., and Sheldon, W , *Lancet*, 1928, *ii.*, 1126.

Other symptoms more definitely referable to the urinary tract may be present. Pain in one or both loins may be complained of, and may be no more than a dull ache, or may amount to attacks of severe colic. Not uncommonly there is a history of frequency, and there may be diurnal as well as nocturnal enuresis. On palpation an enlarged kidney may be felt, and if the obstruction is an intermittent one the swelling may be found to vary in size. In long-standing cases symptoms of renal fibrosis may arise, such as increasing thirst and polyuria, and the blood pressure and blood urea may both be raised. Chronic uræmic manifestations, such as headache, vomiting attacks, giddiness and tremors, may develop, while in others the story of chronic ill health may be brought to an abrupt end by acute uræmia.

Diagnosis. The possibility, and even probability, of some urinary deformity should always be borne in mind when cases of acute pyelonephritis fail to respond properly to medical treatment, and the same may be said of cases which come under observation at a time when the pyuria has already become chronic.

The various methods by which these conditions can be further investigated are largely a surgical matter. Intravenous pyelography is now a recognised and valuable aid in their elucidation, and consists of the intravenous injection of uroselectan (a substance which is secreted in the urine and is opaque to X-rays), followed by an X-ray of the renal tract. The outline of the renal pelvis and ureters is thus revealed, and gross anatomical abnormalities may be depicted. The amount to be injected should be 5 c.c. in infancy, 8 c.c. at two years, 10 c.c. at five years, increasing to 15 c.c. at twelve

FIG. 46. Pyelogram obtained by simple reflux from the bladder, showing a dilated and tortuous right ureter and a right-sided hydronephrosis.

years. Cystoscopy can be carried out in all but young male infants, and may be combined with catheterisation of the ureters with the object both of pyelography and of obtaining separate specimens of urine from each kidney. While reflux of urine does not normally occur from the bladder into the ureters, it may do so when there is chronic pyuria,[1] and advantage can be taken of this to secure a pyelogram. The bladder is first filled with some solution opaque to X-rays (such as sodium iodide 13·5 per cent.), and then the buttocks are raised to allow the solution to run back into the ureters, and an X-ray picture is taken in this position. The illustration shows a pyelogram obtained in this way. The intramuscular injection of dyes such as indigo-carmine and the direct observation through a cystoscope of their excretion from the ureters may also be used to furnish information of the relative function of the two kidneys.

Prognosis. The outlook depends on the nature of the deformity. Those that are so situated as to affect only one kidney may be dealt with surgically with success, but when both kidneys are involved the ultimate outlook is grave owing to the likelihood of progressive renal fibrosis. Eventually infection almost always occurs, and although this may not lead to a fatal issue for some years a slow deterioration of renal function follows, while at any time an exacerbation of infection may lead to fatal suppurative pyelonephritis.

Treatment. Careful investigation must be undertaken to try to discover the nature of the deformity in the hope that surgical relief may be possible, and if it can be ascertained that the lesion is a unilateral one, exploratory operation is justified.

When both kidneys are involved and the renal function is poor, surgical measures are not likely to be successful. Every effort should then be made by medical means to reduce the degree of infection. Treatment by alkalies is generally quite useless, and although extensive trials have been made of hexamine and similar urinary antiseptics, vaccines, and sera, these are mostly without effect. The choice of treatment will lie between the sulphanilamide group of drugs, and mandelic acid.

Sulphanilamide. Sulphanilamide is most effective in a slightly alkaline urine, and it is advantageous to prescribe twenty or thirty grains of alkali a day. This dose is of course not comparable to the larger doses given in the alkaline treatment of acute urinary infections. The concentration of sulphanilamide required for sterilisation of the urine—50 mg. per 100 c.cm. of urine—can usually be obtained without difficulty even in the face of impaired renal function, which gives the drug a definite advantage over mandelic acid. Of the various preparations, sulphathiazole and sulphadiazine have seemed to the author the most reliable, both being well tolerated. It is important to know the nature of the infecting organism, for while the sulphanilamides in general quickly overcome *B. coli* infections, the preparations named above are particularly useful when dealing with that most resistant organism *B. proteus*, and they are often successful against the *Streptococcus fæcalis.* Should they fail, resort may then be made to mandelic acid. The dose of sulphanilamide should follow the table set out on p. 715, and the drug should be continued for a week after the urine has become sterile, otherwise relapses may occur.

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[1] McKhann states that reflux from the bladder occurs in 20 per cent. of cases of chronic pyuria. *Amer. Jour. Dis. Child.*, 1928, **36**, 315.

Mandelic Acid. In 1931 Helmholz introduced the ketogenic diet as a successful means of combating some of the more resistant *B. coli* infections, but the difficulty of getting children to tolerate the diet proved a severe obstacle. Meanwhile Fuller had shown that the good effects of the diet were due to β-oxybutyric acid, but unfortunately this substance is useless when given by mouth as it is quickly oxidised, and none is excreted in the

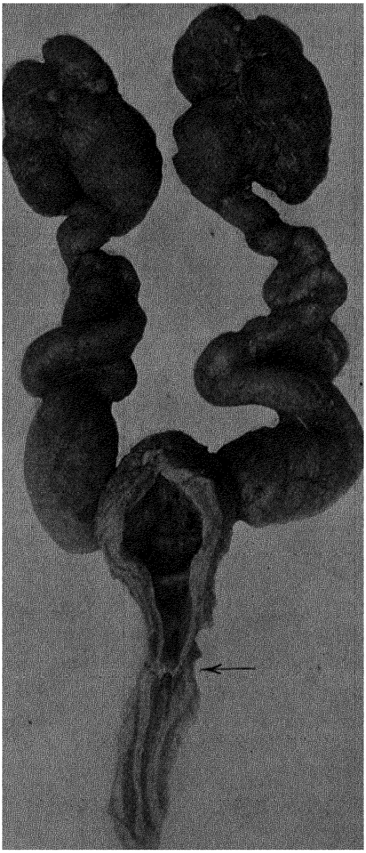

FIG. 47. Congenital mucosal valve (see arrow) in the posterior urethra. Note the hypertrophy of the bladder, the dilatation of the ureters, and the bilateral hydronephrosis. From a boy aged four years.

urine. However, in 1935, Rosenheim demonstrated that mandelic acid acting in an acid urine exerted as good a bacteriostatic effect, and, moreover when given by mouth it is excreted unchanged in the urine.

The two preparations now generally used are ammonium mandelate and calcium mandelate. The latter is insoluble in water, but has the advantage of being tasteless. Ammonium mandelate in the form of the elixir is not unpalatable, and a mixture similar to the elixir in use at Great Ormond Street is as follows [1] :—

℞. Ammonii mandelatis gr. xxvi.
Extracti glycyrrhizæ liquidi m. v.
Elixiris glusidi m. $\frac{1}{5}$.
Aquam ad ℥i.

The dose varies according to the age ; at a year a teaspoonful twice a day may be sufficient, while to a child of ten years a teaspoonful four times a day would be appropriate. It is essential to keep the urine sufficiently acid, the *p*H being maintained below 5·5. At this acidity the urine gives a pink colour with a few drops of methyl red, and a specimen should be tested daily. If the ammonium mandelate does not produce the required acidity an additional acidifying agent must be given, and for this purpose ammonium phosphate may be used in the following mixture :—

℞. Ammonii phosphatis gr. viiss.
Extracti glycyrrhizæ liquidi m. v.
Syrupi limonis m. xv.
Aquam ad ℥i.

Dose : from two to six teaspoonfuls a day, using the methyl red test on the urine as the indicator of adequate dosage. The administration of mandelic acid should be maintained for a week after the urine has become sterile, otherwise a relapse may easily occur.

Although mandelic acid has proved to be a most valuable weapon, it has its limitations. In chronic urinary infection there is often considerable impairment of renal function, and it may then be almost impossible to get a sufficient concentration of the drug in the urine to render it sterile. The type of infecting organism is also important, for while infection with *B. coli* or

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[1] This is similar to the elixir, and contains the equivalent of 1½ grammes of mandelic acid in 1 drachm.

Streptococcus fœcalis responds favourably, those organisms that are associated with an alkaline urine, such as staphylococci, and organisms which break urea down to ammonia such as *B. proteus*, are not destroyed by this treatment.

When there is chronic cystitis, the bladder may be washed out daily with a weak antiseptic solution such as biniodide of mercury 1/20,000.

Congenital Hypertrophy of the Bladder

This rare condition is characterised by hypertrophy and dilatation of the bladder with trabeculation of the mucosa, followed by secondary dilatation of the ureters and bilateral hydronephrosis. The great majority of cases occur in male children.

The condition may come about from various causes. Young[1] has shown that many of the cases are due to the presence of valvular formations of the mucosa in the posterior urethra, situated as a rule in relation to the verumontanum. The valve is so placed that while it obstructs the outflow of urine from the bladder, it allows the passage of a catheter up the urethra into the bladder, and no doubt this has accounted for these valves being for so long overlooked. Other much rarer causes are hypertrophy of the verumontanum, torsion of the penis, and urethral stenosis. In some cases careful search has failed to reveal any cause, and for these a neuromuscular dysfunction at the neck of the bladder has been supposed, and this is to some extent supported by the occasional association with spina bifida. In a slightly different category are those rare instances of dilatation of the bladder and ureters which occur with congenital absence of the muscles of the abdominal wall, for it seems likely that here the dilatation comes about from the lack of a supporting intra-abdominal pressure.

Symptoms. The symptoms date from birth. There is diurnal as well as nocturnal incontinence, and when these occur in association with a constantly palpable bladder they are most suggestive, and at once indicate that the incontinence is no mere functional condition. The enuresis amounts to a continual incontinence, and the child is unable to pass urine in a vigorous stream. The bladder usually extends about half-way between the symphysis pubis and the umbilicus, and feels much firmer than normal.

The destructive effect on the kidneys leads to chronic interstitial nephritis, and in children who survive long enough the symptoms of this condition are superadded to those already enumerated. Thirst is often severe, stunting of growth and rickety deformities become noticeable, and eventually uræmic manifestations appear. Death comes about usually from uræmia, and often takes place within a few weeks of birth, but the child may survive for some years.

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[1] Young, Frontz and Baldwin, *Jour. Urol.*, Baltimore, 1919, 3, 289.

Treatment. Treatment is unsatisfactory. Medical measures are without effect, and surgical interference offers the only chance of success. In a series of 21 children with urethral valves Young was able to record a successful destruction of the valves in 15 of them.

Urinary Tuberculosis

Apart from the miliary form, tuberculosis of the urinary tract is uncommon in childhood. Miliary tuberculosis affects the kidneys in common with other organs, but does not give rise to local signs and need not be further considered.

Caseous tuberculosis of the urinary tract in childhood resembles the condition in adults. The lesion generally begins in one kidney, although bilateral involvement is said to be more common in children than in adults. The process begins as small caseous areas at the apices of the pyramids, and thence gradually invades the kidney substance until ultimately only a shell of renal tissue surrounds a tuberculous pyonephrosis. Meanwhile the infection spreads down the ureter, causing thickening and obstruction, and gives rise to a crop of tubercles in the bladder near the ureteric orifice. At autopsy there is generally an older focus of tuberculosis to be found in some other part of the body.

Symptoms. The onset is insidious. Frequency of micturition occurring both by day and night may be the first warning, and this may soon be followed by recurrent hæmaturia. As the condition progresses there may be attacks of renal colic, and examination may reveal tenderness in one or both loins. The kidney is only likely to be palpable in the late stages. The temperature is usually raised a few degrees, but loss of weight and sweating are late symptoms. The urine is acid, and contains pus and perhaps blood, but is sterile on culture, although tubercle bacilli may be found in the deposit of a 24-hour specimen, or their presence may be proved by inoculating a guinea-pig with the urinary deposit.

Diagnosis. It is most important that the diagnosis should be made early, at a stage when only one kidney is involved. A recent history of frequency or an unexplained hæmaturia should raise one's suspicions, as should a sterile pyuria. At this stage cystoscopy may confirm the diagnosis by revealing tuberculous ulcers in the bladder. Although in long-standing cases an X-ray picture may show some calcification in the kidney or ureter, radiography is not likely to be of help in reaching an early diagnosis.

Treatment. If the infection is confined to one kidney, the kidney and the upper part of the ureter should be removed, and this should be followed by prolonged treatment on general medical principles, including rest, a nourishing diet, and fresh air. When both kidneys are involved the outlook is bad. In addition to general measures a course of injections of tuberculin, using very small doses (see p. 252), is sometimes an advantage. Otherwise treatment is confined to the relief of symptoms.

Perinephric Abscess

Suppuration in the perinephric tissues may occur at any age, and has been recorded in a child of five weeks old. Usually the infecting organism is the *Staphylococcus aureus*, the infection arising sometimes from superficial skin lesions, but at other times the source is indeterminate. As a rule the kidney itself is not involved, although exceptionally the perinephric inflammation may be secondary to the rupture of an abscess from the cortex of the kidney through the renal capsule. As a rule the urine is normal.

Symptoms. The onset may sometimes be sudden with high fever and rigors, but is more usually gradual, and begins with pain in the loin. The pain is persistent and radiates down towards the thigh. There will also be tenderness over the loin, but it may be a week or two before this becomes at all acute. There is likely to be some stiffness of the hip, and the child may lie with the trunk flexed to ease the inflamed part. The tenderness may affect the psoas muscle, causing the hip on that side to be flexed, and in the early stages may give rise to a limp. The temperature is usually high, and may show a daily swing over 3 or 4 degrees. As a rule an abscess gradually forms, causing a swelling which fills out and even bulges the loin, and the overlying skin may become œdematous and red. A blood count shows a high leucocytosis.

Until the signs of an abscess appear the condition may easily be mistaken for tuberculosis of the spine or hip, but the lack of any localised tenderness of the spine, and the full degree of flexion of the hip, help to distinguish these conditions.

Treatment. Fomentations should be applied until an abscess forms, and this should then be incised and free drainage established.

Vulvo-vaginitis

A vulvo-vaginal discharge is not an infrequent complaint in childhood, and is most common between the third and sixth years. The most important infecting organism is the gonococcus, and the condition may be divided into gonorrhœal and non-gonorrhœal types. Analysis of over 1,200 cases by Nabarro and Sharp at the Children's Hospital, Great Ormond Street, showed 20 per cent. to be gonorrhœal.

Non-gonorrhœal Cases. Various factors contribute to simple vulvo-vaginitis. Mere lack of cleanliness of the parts is sometimes the cause, and may be the result of indolence, or to a reticence of the mother in performing a proper daily toilet of the region. An association with threadworms is not uncommon, the worms causing a good deal of irritation which the child attempts to relieve by scratching and rubbing. Other causes of local irritation include constipation, a strongly acid urine, glycosuria, and eczema pudendi. Masturbation is also sometimes a cause, although this habit may equally well be brought on by the irritation of the discharge. Foreign bodies in the vagina such as match sticks, safety pins, etc., are a rare cause, but must be borne in mind. Almost always the general health is much below par and there is often a considerable degree of anæmia.

Local examination generally shows a thin mucoid odourless discharge, although if it has been present for some time it may become purulent and offensive. The labia are reddened and may be stuck together by the discharge. Complications seldom arise. Cystitis and ascending pyelitis may occur, and primary pneumococcal peritonitis is commonly thought to arise by infection ascending from the vulva through the Fallopian tubes.

A bacteriological examination of the discharge must always be made to exclude gonococcal infection. In the simple cases *B. coli*, intestinal streptococci, and diphtheroid bacilli are the usual organisms ; pneumococci and diphtheria bacilli are rare.

Treatment. A careful examination must be made to find any local cause of irritation, always remembering the possibility of foreign bodies in the vagina, and such conditions as constipation, thread-worms, or a highly acid urine must be dealt with. Tonics such as cod-liver oil and malt or Parrish's food should be given for the general debility, and in chronic cases a change of air to the country or seaside will often clear up the discharge when other methods have failed. Local applications are generally better avoided, but the child should be made to sit in a warm antiseptic bath for about twenty minutes twice a day. An iodine bath, adding an ounce of tincture of iodine to three gallons of water, or a potassium permanganate bath, adding enough permanganate to tint the water deep pink, may be used for this purpose. The use of œstrin and sulphapyridine is more fully dealt with under gonococcal vulvo-vaginitis, but both have also been used with success in simple vulvo-vaginitis.

Gonococcal Cases. Gonococcal vulvo-vaginitis may arise from the child sleeping with infected adults, or with other infected children, or from such sources of infection as towels, lavatory seats, or rectal thermometers. Criminal interference is a rare cause.

The onset is usually sudden, and the discharge is from the first thick, yellow, and purulent, and may be blood stained. There may be pain and tenderness during micturition.

Diagnosis. Diagnosis depends on the bacteriological findings. When taking swabs the attendant should wear gloves. The swabs are taken from the vulva or just within the vagina, and on examination will show gonococci generally in pure growth.

Complications. The gonococcus may produce complications by direct spread, and in this way proctitis or urethritis may occur, or the infection may ascend to the uterus and tubes and may thence proceed to gonococcal peritonitis. Complications due to blood stream infection are rare. Arthritis may arise, and generally gives rise to a subacute inflammation of the smaller joints which may easily be mistaken for rheumatism.

Treatment. Gonococcal vulvo-vaginitis is a highly infectious condition, and must be treated with full infectious precautions ; affected children should be isolated, and their management is best left in the hands of experts.

The introduction of œstrin and sulphapyridine have broadened the attack on this disease. Prior to their use, success was obtained by a daily vaginal douche with 1 in 4,000 potassium permanganate, followed by packing the vagina with ribbon gauze soaked in 10 per cent. protargol. The packing was left for twenty-four hours and the whole treatment repeated daily. Generally several months of treatment were required before the condition cleared up.

Sulphapyridine has considerably shortened and simplified the treatment ; the drug should be given orally in full doses according to the age, and the discharge usually comes to an end after a few days. With this treatment, local applications can be confined to a warm antiseptic bath daily, for which purpose a potassium permanganate bath serves very well. Although the infection is often brought promptly to an end, it is wise to insist on swabs being negative over a period of three months after the cessation of treatment before pronouncing a cure.

Œstrin has the property of thickening the vaginal epithelium, making it more resistant to infection. The hormone may be given orally, by injection, or in a vaginal suppository. The oral method is to be preferred, although the dose has to be several times larger than when given by the other routes. Of the various preparations, hexœstrol is the best tolerated; a tablet containing 1 mgm. may be given twice a week, and it may have to be continued over several weeks. A temporary enlargement of the breasts may result.

Gonorrhœal urethritis in male children is very rare. Sharp,[1] in 1927, was only able to collect 10 cases from the literature of the preceding ten years, and added 4 of his own. Treatment consists of giving sulphapyridine by mouth, and this can be combined if necessary with urethral irrigations with 1 in 4,000 solution of potassium permanganate.

Phimosis

Phimosis is so often a reason for medical advice being sought that a few words on the subject will not be out of place. The condition is a common one, and the term indicates a narrowing of the prepuce so that it cannot be retracted over the glans penis.

The degree of phimosis varies, but is seldom, if ever, so severe that the preputial orifice actually obstructs the outflow of urine. Phimosis is commonly blamed for a child straining to pass water, and also for the appearance of herniæ, but this is much more likely to be due to a pin-hole meatus in the glans. When the prepuce cannot be properly retracted, smegma collects beneath it and may cause balanitis, with much irritation. Ulceration may then occur at the end of the prepuce, and the exudate from the ulcer may temporarily seal the preputial orifice, causing much pain and screaming when urine is passed.

Phimosis is relieved by the operation of circumcision. Apart from racial customs, the indications for circumcision consist of inability to retract the prepuce over the glans, or the recurrence of balanitis. Mere length of the prepuce does not call for operation, but a prepuce may be too tight to be retracted and at the same time be redundant in length. Should paraphimosis occur —that is to say, an inability to replace the prepuce after it has been retracted behind the glans—circumcision is also needed.

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[1] Sharp, B. B., *Lancet*, 1927, *ii.*, 653.

CHAPTER XIV

DISEASES OF THE NOSE, THROAT, AND EAR

THE NOSE

Epistaxis

NOSE-BLEEDING is a common occurrence among older children, but is practically never met with in infancy. The causes are various, ranging from trauma, and local infections such as an ordinary cold, measles, or chronic catarrh of the nose, to such general disorders as purpura and the grave blood diseases. The habit of picking at the nose is one of the most common causes. Epistaxis may also be an early symptom of typhoid fever, and troublesome nose-bleedings may occur in children with rheumatic mitral disease. It is also a frequent complaint in children who have been growing rapidly and whose general health has been allowed to get below par, perhaps because of some debilitating illness, or through a combination of overstudy and insufficient rest. The nose-bleeding in these children may be both severe and frequent. Recurrent epistaxis is occasionally due to the presence of telangiectases on the nasal septum.

When epistaxis occurs at night the blood may trickle to the back of the nose and be swallowed, and later on be vomited from the stomach, simulating hæmatemesis. True hæmatemesis in childhood is, however, so uncommon that a history of vomiting blood should always prompt a careful inspection of the nose and naso-pharynx, in case the source of the bleeding lies there.

Treatment. As a rule the bleeding ceases automatically. Observation will generally show that the blood is coming from one side of the nose only, and then by compressing the ala nasi on the affected side against the septum the hæmorrhage can usually be checked in a few minutes. If the bleeding continues, the anterior nares may be plugged with gauze dipped in a 1 in 1,000 solution of adrenalin, and in the most severe cases an injection of hæmostatic serum may be given. When epistaxis is caused by telangiectases, the vessels should be cauterised.

Acute Catarrhal Rhinitis

This amounts to a common cold. The symptoms are too well known to need description, but there are features which apply particularly to children. Owing to the smallness of the parts, obstruction to breathing is much more marked than in the adult, and a cold which does not clear up rapidly may easily sow the seeds of mouth-breathing and persistent enlargement of the adenoids. Also, on account of the relative shortness of the Eustachian tubes, inflammation of the middle ear is a frequent complication, and unless it is promptly treated it may pass on to acute mastoiditis or may give rise to persistent ear discharge. Involvement of the accessory nasal sinuses in childhood is also frequent, and generally has its origin in a " cold ".

It should be remembered that nasal catarrh is one of the early symptoms of measles, and therefore enquiry should be made whether the child has recently been in contact with others suffering from this disease, and examination should include inspection for a rash and a careful observation of the buccal mucosa for Koplik's spots (see p. 679).

Treatment. During an attack of acute catarrhal rhinitis the child should be confined to bed. Too often attendance at school continues, which not only prolongs the individual attack but exposes other children to the infection. An initial purge is beneficial. For older children a nasal spray may be used five or six times a day, spraying with a solution of menthol gr. $\frac{1}{2}$, ol. eucalypti m. 2, and liquid paraffin to 1 oz. In young infants the instillation of two drops of this solution into the nostrils a few minutes before each feed will help to clear the air-passages, and so make breathing easier.

Chronic Catarrhal Rhinitis

A persistent discharge of muco-pus from the nose is not uncommon among poorer children. As a rule the discharge comes from both nostrils, and is usually the outcome of repeated attacks of acute catarrh which have resulted in enlargement of the adenoids or infection of the nasal sinuses. Both these conditions may operate together. In childhood X-ray examination will be necessary to determine the presence of the latter.

When nasal discharge is persistently unilateral the possibility of a foreign body lodged in the nostril should be remembered. The discharge in such cases may at times be tinged with blood, and eventually is likely to become highly offensive. Diphtheritic infection of the nasal passages also gives rise to a discharge which is usually blood stained and may excoriate the skin of the upper lip. In that case bacteriological examination of the discharge will disclose the infecting organism. Nasal polypi are rare in children, but may accompany a discharge from the nose. They arise secondarily to infection of the sinuses.

Treatment. This will depend upon the cause. The removal of infected tonsils and adenoids, of a foreign body, or polypi, come within the province of the surgeon. Inflammation of the nasal accessory sinuses may yield to general hygienic measures, particularly a combination of fresh air at the seaside and good food. A more detailed description of the treatment of sinusitis is given on p. 314. In addition to the eradication of any local focus of infection, an alkaline nasal douche or spray (sodium bicarbonate gr. 20, water to 1 oz.) helps to free the passages of cloying mucus, and may be used in all but infants, in fact older children can quickly learn to use these remedies for themselves. Sprays and douches are to be much preferred to letting the child sniff a lotion up the nose. In infancy, medicated paraffin drops may be instilled into the nostrils as in acute catarrhal rhinitis, and will enable the infant to take food more easily and to breathe more freely.

Allergic Rhinitis (Hay Fever).

Allergic rhinitis is a condition in which the nasal mucosa is sensitised to foreign proteins which reach the patient by inhalation. The protein or proteins concerned in any given case can be ascertained by skin-testing,[1] the offending substance giving an almost immediate wheal.

Hay-fever is an example of seasonal allergic rhinitis, sensitivity being to the wind-borne pollens of grasses and flowers, and it may be pointed out that when skin-testing for these, the tests need only be performed with those pollens which are ripe coincidently with the patient's symptoms. Hay-fever occasionally occurs in quite young children, and is fairly common in those of school age, but is much more frequently met with in adults.

In children, allergic rhinitis is more often perennial than seasonal, the offending proteins being encountered all the year round. House dust, face powder, the dust from horsehair and feathers (the usual stuffing material of bedding and pillows), and the hair of pets and domestic animals, are commonly responsible.

The symptoms consist of a constant and sometimes profuse nasal catarrh, the material discharged being a watery mucus, quite distinct from the thick yellow and even greenish mucopus produced by infective rhinitis and from infected nasal sinuses. The degree of nasal obstruction may be enough to give an

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[1] The sets for skin-testing prepared by C. L. Bencard & Co. Ltd. are very efficient.

"adenoid facies," cause mouth breathing, thicken the voice, and lead the unwary into blaming the tonsils and adenoids, which are often removed, unfortunately with no lessening of the symptoms. Sneezing is a frequent accompaniment, particularly in the early morning. Often these symptoms are associated with asthmatical wheezing, and shortness of breath on exertion.

The diagnosis is apparent when the nose is examined (for which an ordinary electric auriscope serves very well). The mucous membrane appears grossly swollen, and may even hang in greyish polypoid blobs, the space between the septum and the inferior turbinates is much reduced, and what airway remains is filled with watery mucus.

Treatment. During an attack of hay-fever, the child should be rested indoors in a quiet still atmosphere. In both seasonal and perennial types skin-testing should be carried out to detect the foreign substances concerned. It may then be possible to remove these from the environment of the child, for instance, by altering the material used in the bedding, or the child may be desensitised by one or more courses of inoculations. It may be added that the age, and the nervous texture of the patient must be taken into consideration before embarking upon a long series of injections.

An oily spray to the nose to shrink the nasal mucosa is often of benefit, and for this purpose the following prescription has proved effective :—

Mentholis gr. ss.
Olei eucalypti m. ii.
Olei pini m. iii.
Ephedrinæ hydrochloridi gr. v.
Paraffini liquidi ad ℥i.

In young children a drop of this oil may be instilled into each nostril night and morning in place of a spray. Ephedrine by mouth at bedtime is also helpful, giving gr. ¼ up to five or six years, and gr. ½ to older children. It should be given regularly each night for many weeks.

THE NASAL ACCESSORY SINUSES

By JAMES CROOKS, Esq., F.R.C.S.

Affections of the sinuses during childhood have, up to the present day, hardly received the attention they merit. The maxillary sinuses (antra) and ethmoids are present at birth, and the frontal and sphenoid sinuses begin to appear at about three years of age. The antra are probably the most frequently infected, although it must be remembered that diagnosis is easier here than in the other sinuses.

Acute Sinusitis

Acute infection of an air sinus may occur, with closing of the ostium, retention of pus under pressure, and pain and fever, but such a state of affairs is uncommon in childhood. The ethmoids are most likely to be involved, with pain behind the eyes, tenderness above the inner canthus, and œdema of the upper and lower eyelids on the affected side. The condition may easily progress to septicæmia or cavernous sinus thrombosis.

Chronic Sinusitis

Chronic catarrhal or chronic suppurative sinusitis is more frequently met with. In such cases mucus or pus discharges from the sinus into the nose, and gives rise to nasal discharge which may be either continuous or constantly recurring in the form of " colds ". Continual nose-blowing is a common complaint. Often with children the discharge is drawn back into the throat and swallowed, so that the mother may make no mention of it, but it can be seen in the pharynx during the routine inspection of the throat.

The swelling of the nasal mucosa which accompanies sinusitis gives rise to blockage of the nose and mouth breathing. A typical " adenoid facies " may result even though the adenoids have been previously removed. When the adenoids are present they are usually much swollen and heavily infected, and it is often necessary to remove them before improvement in the sinusitis can be looked for. Coughing is another common symptom ; it usually amounts to a frequent clearing of the throat, but may occasionally be so spasmodic as to remind one of whooping-cough. It is by no means uncommon to find that children with pulmonary fibrosis and bronchiectasis have sinus disease too, although it is difficult to say which condition is the primary one. Headaches, chiefly frontal, may occur with inflammation of any of the sinuses. Finally the various evidences of a debilitated state of health are to be expected. A change for the worse in temperament is often apparent, the child becoming morose or ill-tempered and disobedient.

Diagnosis. This will depend in the first place upon the observation of the foregoing symptoms. Swelling of the inferior turbinates, pus in the middle meatus of the nose, and post-nasal discharge must be looked for. Transillumination is of less value in the diagnosis of antrum disease in children than in adults owing to the varying degree of development of the sinuses and the presence of unerupted teeth in the maxilla. It is very desirable to have an X-ray photograph which will show the antra, ethmoids, and frontal sinuses on the one film.

Treatment. This must depend, of course, on the particular sinus involved, and on the nature of the infection, but there are some general factors which apply to all children with chronic sinusitis. Attention to the general health, a generous diet, avoidance of " colds ", and a holiday at the seaside will cure many of the milder cases of catarrhal sinusitis, and ultra short wave therapy may be beneficial. If the sinusitis is accompanied by infection of the tonsils and adenoids they should be removed as a preliminary to other treatment.

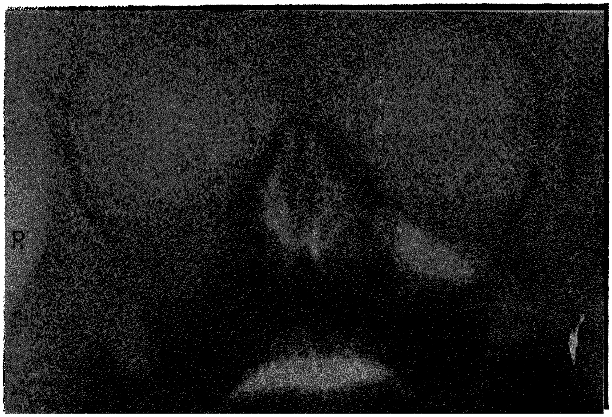

FIG. 48. X-ray of a boy aged six years, showing an infected and opaque right antrum. The left antrum is healthy and translucent.

When infected antra have not yielded to general hygienic measures they should be treated by lavage. This can be done under local anæsthesia (cocaine 5 per cent.) in all but the most recalcitrant children. It will often have to be repeated, perhaps several times. The treatment of ethmoiditis by direct lavage is not practicable.[1]

In more advanced cases of sinusitis, when there is polyposis or fibrosis of the lining membranes, with the formation of pus, more radical means of treatment may be called for. Finally, when there is acute inflammation of the sinuses with retention of pus under pressure, urgent surgical measures will be required.

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[1] By his displacement method, Proltz has instituted a valuable form of treatment for ethmoiditis and frontal sinus infections, and indeed for catarrhal nasosinusitis in general.

THE TONSILS AND ADENOIDS

Simple Hypertrophy of the Adenoids

This is occasionally met with as a congenital condition, when it is likely to give rise to snuffles and mouth breathing. The snuffles and nasal obstruction make it difficult for the infant to suck properly, which may account for the mistaken idea that the mother's milk is unsuitable and so may lead to premature weaning from the breast. The mouth breathing dries the tongue, and mild infections of the mouth, particularly thrush, are then more likely to develop. The obstruction to respiration may sometimes be severe enough to prevent a proper expansion of the lungs, which may be indicated by some recession of the lower intercostal spaces and a sinking-in of the xiphisternum. Attacks of cyanosis may occur, and, if an opportunity for post-mortem examination arises, the borders of the lungs may be found collapsed.

The diagnosis of congenital enlargement of the adenoid pad will be made in the presence of the foregoing symptoms, for it may be pointed out that if the adenoids are hypertrophied sufficiently to need treatment the symptoms are quite pronounced. Snuffles is common in Mongol infants, and may also be one of the early symptoms of congenital syphilis, but in the latter there will generally be other manifestations of the infection, as well as a positive Wassermann reaction, to support the diagnosis.

Treatment. This consists of the removal of the adenoids.

Acute Tonsillitis

Although acute throat infections are common enough in children, the diagnosis of tonsillitis and sore throats is often overlooked because of omission to examine the throat. This is, however, a fundamental part of the examination of children, and if a child is being examined for the first time there are no circumstances which make its omission excusable. Naturally a child often dislikes having his throat looked at, and therefore it is wise to defer it until the other systems have each been examined, which incidentally gives the child time to realise that the doctor is both gentle and understanding. It is a very satisfying accomplishment so to handle a timid or refractory child that even examination of the throat does not upset him. It is essential that the child should be facing a good light, and that the inspection should be made without hurry and with the minimum of fuss so that one view will suffice.

The fact that the tonsils have been removed by no means prevents the throat from becoming inflamed, but in that case a careful examination of the throat is more than ever necessary, for although there may only be a diffuse redness and slight swelling of the fauces and pharynx, the likelihood of complications and sequelæ is as great as though an acute tonsillitis had been present. On this account the term " sore throat " is perhaps preferable to acute tonsillitis, for it includes infections of the throat occurring in the absence of the tonsils.

Etiology. Acute infection of the throat is common at all ages of childhood, and may occur in the youngest infant. Its incidence is highest during the cold and wet months of the winter, although in this country of changeable and uncertain climate the possibility of throat infection must at all times be borne in mind in any ailing child. Sore throats are highly infectious, and in places where children are much together, as in schools or crowded homes, small epidemics are not uncommon. It is doubtful whether sore throats confer any but the most fleeting immunity, for it not infrequently happens that a child has several attacks in the course of a year. The most common causal organism is the streptococcus, usually the hæmolytic streptococcus. Pneumococci, B. Influenzæ, and the organisms of diphtheria and Vincent's infection are less frequently associated.

Symptoms. It is surprising how frequently a child may suffer from quite a severe inflammation of the throat and yet make no complaint to indicate the site of the trouble, and the milder degrees of inflammation would often be missed entirely were it not that the general malaise causes the temperature to be taken, and when a fever of three or four degrees is discovered a routine inspection of the throat is carried out. Vomiting at the onset is usual, and often a cough is complained of, but there may be no loss of appetite nor difficulty in swallowing. The throat appears congested and bright red, the tonsils may be swollen, and the lymphatic glands at the angles of the jaw soon become enlarged and tender.

Follicular Tonsillitis. This represents the most severe form of septic throat. The onset is brisk, and vomiting practically always occurs. The temperature rapidly mounts to 103° or 104° F., the tongue is furred, and the breath soon becomes offensive. Movements of the neck may be painful, and occasionally the neck may be held so stiffly as to suggest meningitis. After a few hours the child may become drowsy or occasionally delirious, and the urine becomes scanty, high coloured, and usually contains albumin and ketone bodies. Constipation is common, but in young children diarrhœa may occur instead. Examination of the throat shows a general reddening and swelling, the tonsils are swollen and acutely inflamed, and their surface is dotted with small yellow beads of pus. These generally remain discrete, but in the most severe instances may coalesce to form a loose yellow membrane which may at times be very difficult to distinguish from diphtheria. The tonsillar lymphatic glands at the angles of the jaw may enlarge to the size of a pigeon's egg, and become tender. In uncomplicated cases the temperature falls to normal within seventy-two hours, and this may be accompanied by heavy sweats. As a rule the tonsils remain swollen for a week or more, while the cervical glands subside in about ten days.

When follicular tonsillitis occurs in a throat that has been repeatedly inflamed and in which the openings of the crypts of the tonsils have already become sealed over with scar tissue, loculi of pus may sometimes be seen just under the surface of the tonsil. Such cases tend to run a more prolonged course owing to the inability of the tonsil to drain properly, the cervical glands are likely to remain enlarged, and other complications such as quinsy, otitis media, or suppuration of the cervical glands are more likely to develop. Haig-Brown in 1886 pointed out that the temperature in acute tonsillitis should return to normal in three days, and that if this does not happen it is probable that some complication is brewing.

Complications may arise as a result of direct spread of infection from the tonsils, or may develop in distant parts of the body after an interval varying from a few days up to three weeks. The first group includes peritonsillar abscess (quinsy), retropharyngeal abscess, otitis media, suppurative cervical adenitis, acute pulmonary infections, and septicæmia. In the second group are included rheumatic polyarthritis and rheumatic carditis, acute nephritis, purpura, and erythema nodosum. Of these, carditis is the most likely to escape detection, and to prevent this the heart should be examined at least once a week for a month after an attack of tonsillitis, otherwise its involvement may pass unnoticed until irreparable damage has been done to the myocardium and valves.

Not uncommonly erythematous rashes closely resembling the rash of scarlet fever accompany a sore throat. When it is remembered that scarlet fever begins as a sore throat and is due to infection by certain specific strains of hæmolytic streptococci, it is scarcely to be wondered at that similar rashes may follow tonsillitis caused by other strains of hæmolytic streptococci, indeed it is likely that such rashes—whether or not followed by peeling of the skin—should be regarded as variants of scarlet fever. There is no doubt that streptococcal tonsillitis, even if unaccompanied by an erythematous rash, is just as infectious and just as likely to give rise to complications as are the cases which develop the typical rash of scarlet fever.

Treatment. It is probable that many of the chronic coughs, nasal catarrhs, ear-discharges, and states of debility which occur so frequently among children of the hospital class would be prevented if more attention were given to the detection of sore throats and more care given to their treatment. It happens too often that no medical care is sought, but the child is allowed to be up and about, thereby delaying his own recovery and acting as a source of infection to others.

The first point in treatment is that the child should be isolated in a room to himself until the inflammation has subsided and until forty-eight hours after the temperature has returned to normal. During this time, he should, of course, be in bed, and on a light diet consisting mainly of fluids, broths, and fruit juices. A brisk purge, such as calomel gr. 1, should be given at the outset. So long as the throat is painful warm fomentations round the neck will give relief, but they should be discontinued as soon as the throat is comfortable for they may incite the cervical glands to suppurate.

Local applications such as paints and gargles are often of value. For young children the throat may be painted twice a day with some antiseptic such as Mandl's iodine paint, but painting the throat should be omitted if it causes much struggling. Children of five years old and upwards can usually manage to gargle quite satisfactorily; gargling with potassium chlorate (gr. 10 in an ounce of water) or with glycothymoline before and after each meal does very well. Potassium chlorate should also be given internally and may usefully be combined with salicylate of soda in the following mixture three times a day :—

Potassii chloratis gr. iii.
Sodii salicylatis gr. v.
Sodii bicarbonatis gr. x.
Syrupi simplicis m. xv.
Aquam ad ℥ii.

After the acute stage is over, tonics such as Parrish's Food or small doses of nux vomica in an alkaline gentian mixture should be given.

The treatment of the various complications is given under the appropriate headings, but mention may be made here of the possible changes in the cervical glands. In some cases they subside shortly after the tonsils have recovered, becoming scarcely palpable. On the other hand there are cases in which the temperature remains high, and one or more glands continue to enlarge, become increasingly tender, and finally soften and fluctuate, the skin over them becomes reddened, and it is clear that an abscess has formed, requiring incision and drainage.

There remain many cases in which the glands take up an intermediate position between these two extremes. Not infrequently a gland at the angle of the jaw swells to the size of a pigeon's egg, but remains firm and is only slightly tender. The temperature hovers round about 100° F. The best course then is to give the gland a chance of slowly settling down by giving it complete rest. Foments should not be applied over it, but the child should remain in bed and the head should be immobilised by placing small sandbags on either side, or a splint may be applied in the same way as when dealing with tuberculous glands in the neck (see illustration on p. 463). Iodine ointment or an iodine paint applied over the gland should be the only local treatment. As a rule the gland slowly subsides in two or three weeks ; should there be no alteration in it after three weeks warm fomentations may be continuously applied with the deliberate object of promoting abscess formation followed by drainage.

Occasionally a mild sore throat is followed by a high swinging temperature lasting for some weeks, while the general condition of the child remains surprisingly good. Usually one or two glands of moderate size can be felt on one or both sides of the neck, and the temperature would seem to be due to toxæmia arising from a small number of organisms which have quickly passed the barriers in the throat and have settled in the glands. The blood picture and absence of swelling of glands elsewhere or of enlargement of the spleen will help to distinguish such cases from glandular fever. I have on more than one occasion seen a temperature of this sort cease abruptly after a dose of streptococcal antitoxic serum. Alternatively the application of foments to the glands for two or three days may enable the infection to be overcome and so bring the fever to an end.

The use of sulphanilamide in sore throats will depend mainly upon the severity of the inflammation. In the milder instances the condition subsides in forty-eight hours, and chemotherapy is not required, but in a severe follicular tonsillitis with high fever and considerable constitutional disturbance full doses should be given for three or four days, not only to cut short the infection but also in the hope of preventing such local complications as suppurative cervical adenitis, quinsy, and otitis media. Sulphanilamide may also be given when, following a sore throat, the cervical glands remain swollen and firm, and are accompanied by a high or swinging temperature.

Quinsy (Peritonsillar Abscess)

Quinsy arises as a complication of acute tonsillitis, and is met with in the later years of childhood rather than in infancy. The accumulation of pus gives rise to a unilateral boggy swelling situated as a rule above the tonsil so as to bulge the soft palate and displace the uvula towards the opposite side. The development of a quinsy is marked by a continuation of high fever beyond the usual two or three days of acute tonsillitis, there is difficulty in swallowing, the speech becomes thick, and there may be considerable pain radiating into the ears or down the neck.

Treatment is surgical and consists of incising the swelling, care being taken that the child is in such a position as not to inhale the pus. A quinsy is likely to recur with later attacks of tonsillitis, and therefore it is generally wise to have the tonsils enucleated later on—at an interval of about four to six weeks after the quinsy has been dealt with.

Retropharyngeal Abscess

This also arises in the majority of cases as a complication of acute infection of the tonsils and pharyngeal tissues, although a tuberculous abscess in the same situation may arise from disease of the cervical spine. The acute variety following sore throat is met with in infants and young children, taking the place of quinsy in older children.

Symptoms. The symptoms are as a rule severe. There is high fever, with vomiting or refusal of food and irritability. The face looks puffy, the cervical glands are generally swollen and tender, and the voice lacks power owing to laryngeal obstruction. The size of the pharynx in infancy is so small that there is little room for an abscess, and the swelling soon gives rise to difficulty in breathing and swallowing, indeed the obstruction to respiration may reach dangerous proportions and cause suffocation. The child usually appears cyanosed, the respirations are rapid and accompanied by inspiratory stridor, and there is likely to be some recession of the lower ribs as a result of the obstructed airway.

Diagnosis. At first sight the distressed breathing with cyanosis and rapid respiration may suggest pulmonary disease, but except for a few stray râles, confirmatory signs of lung disease cannot be made out. A history of a day or two's illness with stridor and laboured respiration should always call to mind the possibility of a retropharyngeal abscess, and should lead to a digital examination of the pharynx. This should be carried out speedily but dexterously by passing the forefinger over the tongue and feeling the posterior pharyngeal wall. Normally this is a firm structure, the mucosa being closely bound to the cervical spine, but a retropharyngeal abscess gives rise to a soft boggy swelling, the upper and lower limits of which can usually be defined. Actually the abscess is due to suppuration beginning in a gland on one or other side of the pharynx, but as it enlarges it spreads across and bulges the whole posterior pharyngeal wall, so that it appears to be situated either in, or close to, the midline. It is often visible at the back of the mouth, but may be lower down out of sight opposite the larynx.

Treatment. A retropharyngeal abscess should be looked upon as one of the emergencies of childhood, a good prognosis depending on early diagnosis and prompt treatment. Evacuation of the pus brings quick relief, and thereafter recovery is usually uneventful. When opening the abscess the child should be so placed as to minimise the risk of pus being inhaled into the chest. It is usually advisable to avoid a general anæsthetic.

Chronic Infection of the Tonsils and Adenoids

Chronic disease of the tonsils and adenoids is usually the result of repeated attacks of acute infection, although these may often have been so mild as to have passed unnoticed. Although chronic tonsillitis may be met with at all ages it is most common in children over the age of two years. Overcrowding, bad ventilation, infected atmospheres, and defective dietary are factors which promote acute inflammations of the throat and so assist in causing chronic disease of the tonsils and adenoids.

The *symptoms* may best be considered as local and general.

Local Symptoms. The local symptoms are due in part to hypertrophy and in part to infection. Hypertrophy of the adenoids gives rise to a typical facies—the expression is dull, the nostrils are small and the alæ nasi are indrawn, the mouth is constantly open, and when the condition has been present since infancy the palate may be narrow and high. There is likely to be a history of snoring, and the voice may have a nasal character. Mere enlargement of the tonsils without coincident infection often produces no symptoms, but if they are very big they may cause retching and vomiting and may thereby account for refusal of food.

The local symptoms due to infection include persistent enlargement of the glands at the angles of the jaw, chronic nasal catarrh, and otorrhœa. Recurrent exacerbations of infection are likely to arise in chronically infected tonsils, and at such times the glands in the neck will swell and the local catarrhal symptoms will increase. Chronic tonsillitis may sometimes account for a child having unpleasant breath.

General Symptoms. The general effects of chronic tonsillitis may account for much ill-health. Some degree of secondary anæmia is usual, and at times there may be a general reaction of the lymphatic tissues as shown by small chains of glands in the neck, slight enlargement of the axillary glands, and a just palpable spleen. The temperature is often raised one or two degrees, and may continue so for weeks at a time. It may be raised throughout the twenty-four hours or only in the evenings, but in either case, once it has been discovered, it is likely to cause much alarm to the parents, who suspect tuberculosis or other serious illness.

A trace of albumin in the urine is not uncommon, and in older children may be associated with a general lowering of postural tone. The stomach and intestines often bear the brunt of chronic naso-pharyngeal infection, largely because of the constant swallowing of muco-pus. The usual effect is to produce a catarrhal gastritis, which leads to a severe loss of appetite and consequent malnutrition. Not infrequently there is a history of the child vomiting first thing in the morning, the vomit consisting mostly of mucus which has collected in the stomach overnight. A similar state of affairs may affect the intestine,digestion becomes impaired, and the stools are likely to be undigested, offensive, and may contain an excess of mucus. Constipation is not uncommon. At times, in association with renewed infections in the throat there may be sharp attacks of abdominal pain of a colicky nature, together with vomiting and some tenderness localised in the region of the umbilicus. Such attacks may closely simulate appendicitis; in fact, when the diagnosis of recurrent appendicitis is raised a thorough examination of the throat should be carried out, since removal of infected tonsils and adenoids may lead to a cessation of the abdominal attacks. The presence of threadworms is sometimes attributed to naso-pharyngeal sepsis, but if there is any relation between the two it is an indirect one, the throat infection contributing to the mucous catarrh of the bowel which in its turn tends to keep up the presence of the worms.

Chronic hypertrophy of the tonsils and adenoids, particularly of the latter, often gives rise in young children to severe deformity of the chest-wall owing to the defective entry of air. There may be considerable recession of the lower intercostal spaces with relative prominence of the sternum, in other cases the xiphisternum becomes indrawn, forming a deep hollow at the lower end of the chest. Chronic infection of the trachea and bronchi is common, the tracheitis accounting for the constant hacking dry cough from which these children so commonly suffer. Repeated attacks of bronchitis are likely to occur, especially in the winter, and may progress to broncho-pneumonia. Although naso-pharyngeal infection plays but an insignificant part in true spasmodic asthma, the recurrent attacks of bronchitis may at times be accompanied by wheezing and laboured breathing—attacks which are often called asthma although the name "asthmatic bronchitis" more correctly describes them (see p. 347).

Nervous disturbances of one sort and another are often attributable to persistent throat infection. Frontal headache may be complained of by older children, and giddiness and fainting attacks may also occur. Many of the nervous disturbances result from interference with sleep, caused no doubt in part by the obstructed airway. Restlessness and difficulty in getting off to sleep may be the chief trouble, or night terrors may occur, or there may be sudden attacks of nocturnal croup (laryngitis stridulosa). Bed-wetting may be another troublesome symptom. Facial grimacing, twitching, and other varieties of tics may be further evidence of the harmful influence of throat infections on the nervous system. Although epileptic children may also suffer from diseased tonsils there is seldom any close relation between the two; this point is mentioned here because one not infrequently sees epileptic children who have undergone tonsillectomy without any lessening of the fits.

Relation to other Diseases. Clinical experience goes to show that there are a group of diseases, such, for example, as chronic œdematous nephritis and rheumatoid arthritis, which are associated with foci of persistent infection or of toxic absorption, and in childhood the naso-pharynx is the most common site of such chronic infection, but although removal of the infected tonsils may have a beneficial influence on the course of these diseases this is by no means always the case. While there can be no doubt of the relation that exists between *acute* tonsillitis and the onset of acute rheumatism—whether it affects the joints, the heart, or appears as chorea—the relationship between *chronic* naso-pharyngeal infection and acute rheumatism is less clear, and although large numbers of children have had their tonsils removed in the hope of diminishing the incidence of rheumatism, there is little evidence to show that this hope has been fulfilled. The present-day attitude may be summarised by saying that the tonsils should not be removed just because a child is rheumatic, but whether the child be rheumatic or not the tonsils should be removed if there is evidence that they are the seat of chronic infection and are interfering with the general health.

Appearance of the Throat. The appearance of the throat varies a good deal, and the size of the tonsils affords little indication of the degree of their infection. In some cases the tonsils may practically meet across the midline, and the symptoms that result are then due as much to obstruction as to infection, although in favour of the latter it must be said that there is usually some enlargement of the cervical glands when the tonsils are of this type. On the other hand, the tonsils may be much reduced in size owing to fibrosis, and may be buried behind the anterior pillar of the fauces, but even so the persistent firm enlargement of the tonsillar glands at the angles of the jaw stands as evidence of their unhealthy state. Further evidence of chronic infection may sometimes be obtained from the surface of the tonsil being smeared over with a thin membrane of scar tissue which obliterates the crypts and makes the surface of the tonsil appear continuous with the faucial pillars. In other cases there may be one or more dilated veins coursing down the pillars.

Treatment. The treatment of chronic infection of the tonsils and adenoids by medical means is as a rule unsatisfactory, for by the time these tissues become the seat of chronic inflammation medicines and gargles cannot reach the diseased area owing to it being shut off by fibrous tissue. The only satisfactory treatment consists of the complete removal of the tonsils and adenoids. The choice of method must lie with the surgeon, but whatever method is used the value of the operation lies in its thoroughness, and anything less than a complete enucleation leaves the child in a worse state than if no operation had been undertaken. Tonsil remnants are inevitably heavily scarred, the normal drainage system of the crypts is no longer effective, and the child is left with a chronic septic focus which may at any time be the starting point for an attack upon some other part of the body. One sometimes hears of children having to have their tonsils removed a second time, because, forsooth, they have "grown again." In actual fact this is nothing but a reflection on the skill of the first operator.

However well the operation is performed, complications sufficient to keep the child in bed occur roughly in 1 out of every 200 cases. The most common is earache. In others the operation may be followed by bronchitis, broncho-pneumonia, or occasionally by a pulmonary abscess due to the inhalation of septic material. Acute hæmorrhagic nephritis may be a direct sequel, while in children already the subjects of rheumatism a sharp exacerbation of rheumatic symptoms occasionally follows. In a small number septicæmia occurs through direct infection of the blood stream from the raw surface of the throat.

Another complication which has on rare occasions followed tonsillectomy, and which may also follow other inflammatory conditions in the region of the throat such as tonsillitis, mastoiditis, or suppurating glands, is a forward dislocation of the atlas on the axis. The head is held forward and tilted to one side; movement of the neck is strongly resisted, and the child may support the head by cupping the chin in his hands. A lateral X-ray will immediately confirm the diagnosis.

Tuberculosis of the Tonsils

This condition is more fully dealt with under diseases of the lymphatic glands. Infection of the tonsils by tubercle bacilli may either arise from the child swallowing infected food, particularly milk, or may occur by inhalation. The onset of the infection is as a rule quite insidious, and usually the first thing to be noticed is enlargement of the glands in the neck. Although tuberculous cervical glands are almost always infected from the tonsils, the tonsils themselves may not appear enlarged or otherwise unhealthy; in fact it is impossible to recognise tuberculous tonsils *in situ*, but confirmatory evidence of their condition can be obtained after their removal by making a histological examination of them.

AFFECTIONS OF THE EAR

Acute Otitis Media

Acute otitis media is particularly common in young children, largely on account of the frequency of throat infections at that age. In infancy, moreover, the Eustachian tube is relatively shorter and more horizontal than in later years, making infection of the ear from the throat an easier matter.

Symptoms. In some cases the symptoms clearly point to trouble with the ears. The child is taken suddenly ill with a high temperature, and indicates the source of his pain by rolling his head from side to side on the pillow or by frequently pulling at his ear. In others the symptoms may be only general, consisting of fever and great irritability. The crying is usually more than mere peevish whining, and may amount to bouts of intense screaming. Crying due to otitis media is often particularly noticeable at night-time, and a history of this should always warn the physician to examine the ears. During the attacks of pain the baby may curl himself up, but more often becomes stiff and arches his back. Refusal of food is another symptom at times very prominent, and vomiting is common at the onset and may continue until the ear discharges. Not infrequently in infancy the vomiting is accompanied by diarrhœa and a quick loss of weight, and the gastro-intestinal symptoms may be so prominent that unless the physician is on his guard he may easily overlook the initial trouble in the ears. This is illustrated in the chart (Fig. 49) of an infant aged five months who experienced two attacks of otitis media within a month. On each occasion the weight fell steeply as soon as the temperature began to rise, and continued to fall for some days until the ear discharged. In other cases there may be symptoms of meningeal irritation, as indicated by stiffness of the neck or actual head-retraction and Kernig's sign, and convulsions may also occur, making a clinical picture still more suggestive of meningitis. If real doubt exists a lumbar puncture should be done, for in meningeal irritation (meningismus) the cerebro-spinal fluid may be under pressure but is otherwise normal. Reflex disturbances such as meningismus and diarrhœa become less frequent after the period of infancy.

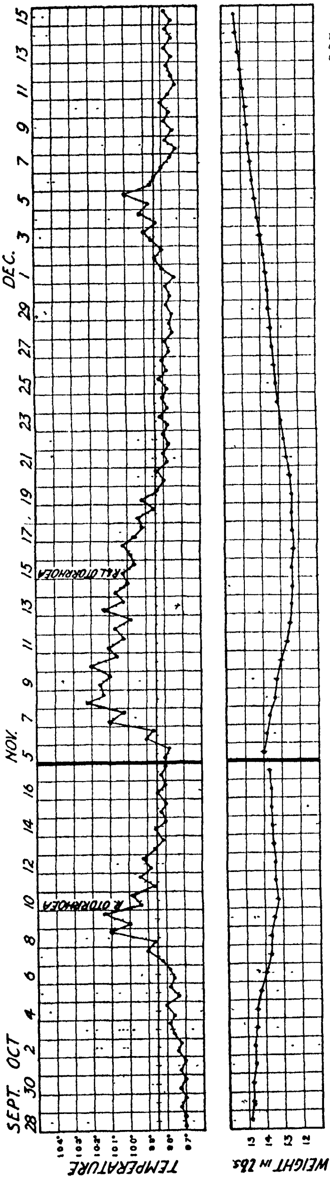

FIG. 49. Temperature and weight chart of an infant aged five months. The clinical history is given in the text on page 325.

A proper examination of the ear is, of course, essential. An electric auriscope gives a good view, but in an infant the smallness of the parts may make examination of the tympanic membrane difficult. In early cases there may be little except loss of the light reflex, while in others the drum may appear dull, with dilated vessels running along the handle of the malleus, or the membrane may be yellow and bulging. The posterior wall of the external auditory meatus is often an unnaturally bright red colour.

Treatment. The local treatment of the ear will depend on the extent of the disease, and is a matter for an aural expert. When the drum is under tension and bulging, puncture of the membrane is often followed by dramatic relief of the symptoms, and as a rule the incision should be carried out at once, for it is much better to do this than allow the drum to burst of itself, since in the former case healing may be expected in a week or two, while in the latter, as a result of the perforation being inadequate in size, a troublesome otorrhœa may persist for months. It may happen that the temperature remains high and the child continues to be obviously ill after a paracentesis. It is then likely that the inflammation has progressed to mastoiditis, and unless this is given prompt surgical treatment further complications such as thrombosis of the lateral sinus or meningitis may develop.

When it is decided to defer paracentesis, or while waiting for an aurist's opinion, considerable relief of pain may be brought about by applying warm foments over both ears. In an infant it is important to see that the foments are warm rather than hot, so as to avoid any risk of scalding, and they should be large enough to cover the whole ear easily ; they are best kept in position by a large pad of wool and a closely-fitting bonnet. Foments are particularly useful at night-time, and they should always be applied to both ears. Additional relief is afforded by instilling drops of warm glycerinated carbolic acid (2½ per cent. in glycerin) into the ear every four hours. A small pipette should be used for this purpose, and the child should lie on the opposite side for a few minutes to allow of penetration of the glycerin. As a further measure of relief small doses of chloral should be given, such as 1 gr. every four hours to a child of a year.

When discharge from the ear has already appeared, the ear should be gently syringed with a warm alkaline lotion (sodii bicarb. gr. 10, sod. chlor. gr. 10, water 1 oz.), repeating this as often as is necessary to keep the parts clean. The best and safest syringe is a rubber rat-tailed syringe, which can be readily sterilised by boiling. After syringing, the ears should be dried out with sterile cotton wool ; one drop of rectified spirit is then instilled as a dehydrant, or a drop of 10 per cent. Protargol may be used instead as an antiseptic.

There is no doubt that the use of sulphanilamide in acute suppurative otitis media reduces the incidence of mastoiditis, and therefore is to be recommended. The reader may be reminded of the part played by ear infection in the parenteral diarrhœa of infants ; otitis media at this age is as deserving of chemotherapy as it is in older children.

Chronic Otorrhœa

As a rule chronic discharge from the ear is the outcome of an attack of acute otitis media in which the tympanic membrane has been allowed to rupture spontaneously, and is often associated with disease of the mastoid. A proper examination of the ear after a preliminary cleansing is essential, for it may be found that the discharge is derived from the external auditory meatus and may be due to a weeping dermatitis or possibly a foreign body.

Treatment. The first essential in the treatment is to exclude any focus of infection in the nose or throat, for if such be present no amount of local treatment will cause the ear to heal. Infected adenoids, septic tonsils, and diseased nasal accessory sinuses are the main causes of chronic otorrhœa, and treatment cannot be considered as adequate until the tonsils and adenoids have been removed, and an X-ray has been taken of the sinuses, and any disease it reveals treated.

Finally otorrhœa may persist after the above measures have been carried out. In such cases the mastoid antrum and cells are diseased, and a conservative mastoid operation must be performed. The radical mastoid operation will only be required when the ears have been so neglected that much destruction of the drumhead has taken place and the middle ear is filled with granulations.

Chronic otorrhœa is occasionally due to tuberculous disease of the middle ear, a condition which is said to be most common during the first two years of life. The disease gives rise to progressive disorganisation of the middle ear, and the facial nerve may become involved. Treatment should consist of a radical mastoid operation.

Acute Mastoiditis

Acute mastoiditis may be met with at any age, even in infants of a few months. It arises as a complication of acute otitis media, generally after an interval of a few days, but may develop almost simultaneously with the ear infection and so may be present before there is any ear discharge. If discharge is already present it often lessens or ceases when the mastoid becomes involved. The temperature is raised to 101° to 104° F., and generally there is severe pain, which an infant may indicate by screaming, and rubbing or pulling at his ear. Pain is, however, not always present. Vomiting is common, there may be diarrhœa, and the infant may appear drowsy and pale. Local signs consist of tenderness and swelling. The tenderness is usually localised over the mastoid process, and pressure in this region may cause the child to wince or cry out, but when the temporal bone is compact and the mastoid air-cells are few or absent, firm pressure or percussion may be necessary to elicit it. Swelling generally develops behind the ear, filling the post-auricular sulcus, but it may be absent even when there is no doubt of the mastoiditis.

Treatment. The treatment is surgical, consisting of immediate opening and draining of the infected bone. There should be as little delay as possible in this, as there is always the risk of further extension of infection causing such complications as thrombosis of the lateral sinus, meningitis, or an intracranial abscess. Although sulphanilamide may be given in order to cut short the infection and lessen the likelihood of further complications, it cannot take the place of operation.

CHAPTER XV

DISEASES OF THE RESPIRATORY SYSTEM

CROUP

CROUP is a household word used to describe the various stridors in childhood, and arises from a variety of causes, but of itself does not constitute a disease. As a rule croup is due to laryngeal disorders, and in reviewing its various causes a description will be given of the affections of the larynx in children.

Congenital Laryngeal Stridor

This is due to stenosis of the larynx, and is brought about by a narrowing of the rima glottidis, the aryteno-epiglottidean folds being approximated so as to give the epiglottis an appearance of being pinched from side to side.

Symptoms. The main symptom consists of a crowing or stridulous sound accompanying inspiration. The stridor begins at birth or within a week or two, and at first may be heard during the night as well as the day. The obstruction is sometimes great enough to lead to deficient aeration of the lungs with considerable indrawing of the xiphisternum and lower ribs. In milder cases the stridor is only heard when the breathing is exaggerated, as for instance, when the infant is crying or is excited. The early onset of the stridor, and its persistence over several months with, as a rule, but little disturbance to the health of the infant, generally makes diagnosis an easy matter.

The majority of cases recover, the stridor gradually lessening and finally disappearing towards the end of the first year. The crowing first passes off at night-time and when the infant is breathing quietly, and only in the more severe cases does it persist into the second year. The chief danger lies in acute pulmonary infections such as bronchitis or pneumonia, for the laryngeal deformity acts as an additional handicap which may just turn the scale against the infant. Recovery is generally thought to be due to a gradual correction of the deformity taking place as the parts increase in size with the child's growth.

Treatment. The deformity cannot be influenced by treatment, but care must be exercised to prevent ordinary coughs and colds, particularly by avoiding contact with children or adults who have upper respiratory catarrhs. The occurrence of a simple laryngitis may rapidly increase the dyspnœa and even necessitate tracheotomy.

Acute Laryngitis

Acute laryngitis is nearly always secondary to infection in the naso-pharynx. It also occurs during the early stages of measles, and may be present at the onset of whooping-cough.

The chief symptom consists of hoarseness or loss of the voice. The temperature is raised three or four degrees, and older children may complain of soreness of the throat. When laryngitis arises in infancy obstructive symptoms such as cyanosis and retraction of the chest wall are to be expected on account of the smallness of the parts and the ease with which the airway becomes narrowed by laryngeal œdema, indeed at times there may be a real danger of suffocation, and it may be necessary to give relief by tracheotomy. Such severe symptoms are, however, uncommon in simple catarrhal laryngitis, and as a rule within two or three days the temperature returns to normal and the voice regains its power. Urgent symptoms are more likely to arise in the acute laryngitis which follows the inhalation of some irritant, such as inhaling steam from the mouth of a kettle.

Diagnosis. The diagnosis of acute laryngitis is as a rule easy. The most important condition from which simple laryngitis must be distinguished is laryngeal diphtheria. The laryngitis of measles is likely to be accompanied by catarrh of the nose and eyes, and the presence of Koplik's spots in the mouth will confirm the diagnosis. It is more difficult to exclude whooping-cough because the laryngitis occurs during the early catarrhal stage before the whoop develops. A history of contact with another case would act as a warning.

Treatment. Even in mild cases the child should be nursed in bed until the temperature has fallen. When coughing is troublesome the use of a steam kettle will often give relief, and the steam may with advantage be impregnated with Friar's Balsam, allowing one teaspoonful to one pint of warm water. A free action of the bowels must be obtained, and the following sedative mixture may be used to relieve the cough, the dose being suitable for a child a year old.

℞ Syrupi scillæ m. iiss.
Syrupi tolutani m. iiss.
Syrupi pruni virginianæ m. iiss.
Tincturæ camphoræ compositæ m. iiss.
Glycerini m. x.
Aquam camphoræ ad ʒi.

Should œdema threaten, or the child become cyanosed, warm fomentations should be frequently applied to the throat to ease the breathing. In small infants one must be prepared to perform intubation or tracheotomy if cyanosis progresses.

Diphtheritic Laryngitis

This condition is considered more fully under Diphtheria, but is referred to here because of the great importance which attaches to its early recognition. As a rule the development of membrane in the larynx follows upon faucial diphtheria, and if examination of the throat shows any traces of membrane a laryngeal stridor can have but one interpretation. There may be a history of exposure to infection; or there may be a sanious discharge from the nostrils indicating nasal diphtheria. The temperature is generally high, the glands in the neck are swollen, the breath may be offensive, the face has a bloated appearance, and the child looks exceedingly ill. Although a throat swab should be taken to confirm the diagnosis, if there is any reason to suspect that the laryngitis is diphtheritic no time should be lost in giving anti-diphtheritic serum.

Syphilitic Laryngitis

This is also considered under Congenital Syphilis. Suffice it here to say that it is an early symptom, occurring generally under three months of age and usually in association with snuffles, and giving rise to hoarseness of the cry.

Laryngismus Stridulus

This condition has already been mentioned as one of the nervous manifestations of rickets, and in company with convulsions and tetany completes the clinical triad spoken of as "spasmophilia."

It may be mentioned again here that laryngismus stridulus is an early symptom of rickets, occurring between four and twelve months of age. Each attack consists of a momentary spasmodic closure of the glottis followed by a crowing inspiration. The whole attack lasts only a few seconds, but may recur many times in a day. The prognosis is good, the attacks clearing up quickly under anti-rachitic treatment.

Laryngeal Spasm

Somewhat similar attacks of laryngeal spasm have been recorded in the first few weeks of life. In the attacks the infant makes every effort to breathe, but fails to fill the lungs, becoming progressively cyanosed and seeming in imminent danger of death. During the attack, breathing should be stimulated by a cold sponge applied to the neck.

Indigestion has seemed at times a factor. Similar attacks occur occasionally in early infancy when the frænum of the tongue is unduly long, allowing the tongue to fall back and obstruct the air-way.

Laryngeal Papilloma

This is an uncommon cause of stridor. The condition generally arises soon after infancy, the symptoms consisting of hoarseness and feebleness of the voice, stridulous breathing, and attacks of cyanosis and shortness of breath. The growth—or growths, for they are generally multiple—slowly enlarge, and the symptoms gradually increase in severity. The diagnosis can only be made by laryngoscopy. The treatment is surgical and consists of the removal of the tumours.

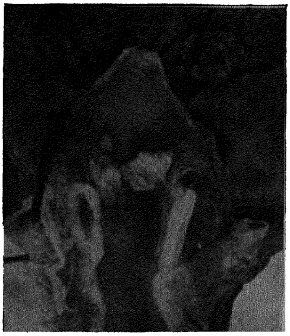

FIG. 50. White papillomatous masses on the vocal cords. From a child aged fourteen months.

Laryngitis Stridulosa

This affection is confined to childhood, and is characterised by attacks in which the child wakes up at night with laryngeal spasm accompanied by dyspnœa and stridulous inspiration. It must not be confused with laryngismus stridulus.

Laryngitis stridulosa may occur at any age from two to ten years, but is most common between three and six years. The sexes are equally affected. Often the children are of a nervous, excitable disposition, but there are additional factors at work as well—for instance some children only experience the attacks when they have a cold or are feverish, in others constipation plays a part. Other attacks are provoked by large and indigestible suppers, as in the case of a girl of five years whose attacks ceased when her bedtime meal of fried fish was stopped.

In a typical attack the child goes to bed seemingly well, but wakes up two or three hours later with urgent dyspnœa. Sitting up in bed struggling for breath, sweating, cyanosed, and making loud inspiratory stridors, the terror of the child is pitiful. The attack gradually subsides in an hour or so, the child drops off to sleep, and in the morning the terror by night is forgotten. There may be slight residual bronchitis for a day or so.

The prognosis for the individual attack is good, the child invariably recovering even when the symptoms are so severe as to seem fraught with danger. The attacks are very likely to recur, perhaps as a rapid burst for a few nights in succession, or a few months may elapse between each attack.

Treatment. During an attack relief may be given by applying warm fomentations or hot flannels over the larynx, and by giving a warm drink. The child needs to be reassured and soothed.

The prevention of attacks demands in the first place a thorough overhaul of the daily routine. The last meal should be at tea time, and should consist only of plain fare. In some cases the attacks have seemed to be associated with an obstructed airway by enlarged tonsils, and have ceased when these have been removed. A course of sedative drugs at bedtime is beneficial. For a child of five years, chloral gr. 3, luminal gr. $\frac{1}{4}$, or phenazone gr. 5, may be given.

Foreign Body in the Larynx or Trachea

Foreign bodies in the respiratory passages are more common in children than in adults, owing to their habit of putting things in their mouth. When the foreign body enters the larynx a violent fit of coughing is provoked and the child may choke and become cyanosed. Should the foreign body be large enough to block the airway, death will of course rapidly ensue from suffocation unless a tracheotomy is immediately undertaken, but more often the foreign body allows of the passage of air beyond it, and this causes a stridor which may sometimes be loud enough to be heard across the room, or may be so quiet that it is only heard by listening close to the child's mouth. A foreign body in the larynx will cause hoarseness or loss of the voice. When the foreign body is metallic, it may show on an X-ray film, but a negative X-ray does not exclude the presence of a foreign body. If suspicion of a foreign body is entertained, an expert endoscopic examination should be undertaken. Removal of the foreign body should likewise be left in the hands of an expert.

Stridor may finally be due to pressure on the trachea from without. One of the most important causes in young children is a retropharyngeal abscess (p. 319). Stridor from compression of the trachea by congenital goitre has been recorded; and during the first year of life it may be associated with enlargement of the thymus (p. 483). Also at this age stridor may be due to the pressure of enlarged mediastinal glands, which are usually tuberculous. The following case may be cited as an instance. A baby, six months old, had had for six weeks stridulous respirations with much recession of the lower ribs. There was a history of contact with an adult suffering from phthisis. A tuberculin skin reaction was strongly positive, and an X-ray film showed the presence of enlarged glands at the roots of the lungs. The stridor continued for several months, but eventually cleared up as the glands healed.

TRACHEITIS

Tracheitis is probably more common than is generally realised. Acute inflammation of the trachea is but an item in the spread of infection from the throat to the bronchi, and is sometimes a troublesome complication of influenza. Many of the coughs in childhood which are unaccompanied by physical signs are probably due to chronic tracheitis. As a rule the cough is hard, dry, and often worse just after the child has gone to bed. In some cases there is clear evidence of associated naso-pharyngeal infection, but not in all. A cough of the type just described is best dealt with by rubbing the chest at night-time with a stimulating liniment (turpentine liniment is as good as any), and giving some such cough mixture at bedtime as the following:—

℞ Potassii citratis gr. v.
Tincturæ camphoræ compositæ m. v.
Syrupi tolutani m. v.
Syrupi scillæ m. v.
Glycerini m. x.
Aquam ad ℥i.

BRONCHITIS

Acute Bronchitis

This is common at all ages, but particularly so at the extremes of life. The exciting cause is acute bacterial invasion of the bronchial mucosa, the infection spreading down from the nose and naso-pharynx. Of the infectious fevers, measles, whooping-cough, and influenza are nearly always accompanied by acute bronchitis. In young children there may be additional factors; for instance, rickets is a predisposing cause, partly owing to the softness of the thoracic cage with consequent poor expansion of the lungs, and partly because of the increased susceptibility of rickety children to infections in general. Many of the babies who are said to "cut their teeth with bronchitis" are also the subjects of rickets. Congenital heart disease also renders a child more prone to bronchitis.

Symptoms. The onset is brisk, the first symptom being a cough which continues throughout day and night, disturbing the sleep. There may be some pain beneath the sternum. The cough, which at first is dry, soon becomes loose and moist, but is not accompanied by any visible sputum, since children invariably swallow it. The face is often flushed, the temperature rises three or four degrees, and in infants may reach 104° F., and the respirations rise to 30 to 60 per minute.

As the inflammation passes to the smaller tubes the child becomes cyanosed. The alæ nasi dilate, and the accessory muscles of respiration may be brought into play. In young infants the exudation may block the smaller tubes, leading to collapse of the portions of lung beyond, and increasing the degree of cyanosis, indeed it is often impossible to say exactly when these infants pass from bronchitis to broncho-pneumonia. While in older children bronchitis is seldom a dangerous illness, in infancy it may easily prove fatal.

The signs are the same as in adults. The percussion note is not altered, but on auscultation numerous adventitious sounds are to be heard over the lungs. Over the upper part of the chest the sounds may be dry and sibilant, while moist sounds will tend to collect at the bases. When areas of lung become collapsed the crepitations may take on a clicking character indistinguishable from those heard in broncho-pneumonia.

Diagnosis. As a rule the diagnosis is straightforward. The possibility that the illness marks the onset of measles or whooping-cough must always be borne in mind. In young infants there may be great difficulty in distinguishing bronchitis from pneumonia, and one has to be guided as much by the general appearance of the child as by the signs in the chest. The temperature is likely to be higher in broncho-pneumonia, and the child is more listless and irritable, and looks obviously more ill. There is a greater degree of cyanosis, and the pulse and respiration rates are more rapid. It must also be remembered that miliary tuberculosis of the lungs may only present the signs of bronchitis, but the weakness and rapid wasting, the dyspnœa and cyanosis out of proportion to the signs, and the high swinging fever will point to tuberculosis. Lastly, attacks of spasmodic asthma in infancy may closely resemble bronchitis, but the frequent recurrence of the attacks, their rapid onset and as brisk disappearance, the absence of fever, and the possible association with eczema, help to distinguish them.

Course and Prognosis. Simple bronchitis should clear up in a week or ten days, but the uncertainty at first of telling whether the condition will pass on to broncho-pneumonia must make the prognosis guarded. The outlook is worse in premature or feeble infants, and in those with rickets or congenital heart disease.

Treatment. The child should be nursed in a warm room at an even temperature, but with an adequate supply of fresh air. Clothing should be of loose woollen material. The diet should be mainly of fluids, which need not be restricted. Milk, milk puddings, fruit and fruit juice, custards, broths and jellies may be given. For the infant who is artificially reared, the milk mixture should be reduced in strength, though not in amount, so long as the temperature is high. At all ages it is well to begin treatment by giving a purge.

A steam kettle may sometimes be applied with advantage, but its uses must be clearly understood. It should only be used when the cough is frequent and dry, but should be discontinued as soon as the sounds in the chest become moist. The kettle should stand out in the room at some distance from the child, its function being merely to moisten the atmosphere, and under no circumstances should a steam tent be used. It is as well to impregnate the steam with some soothing preparation such as the addition of one teaspoonful of Friar's Balsam to one pint of water.

The application of stimulants to the chest wall as a means of counter-irritation finds its greatest value in young children, owing to their relatively large skin surface. The chest should be rubbed morning and evening with some stimulating liniment such as Lin. Terebinth. The common household remedy of camphorated oil is also useful, although not so stimulating. Heavy applications such as stupes, poultices and plasters are unnecessary in simple bronchitis, and in young infants they tend to hamper the respiratory movements.

During the early stage when the cough is dry and irritating a sedative mixture such as the following will be found useful :—

℞ Potassii citratis gr. v.
Tincturæ ipecacuanhæ m. v.
Tincturæ camphoræ compositæ m. v.
Syrupi simplicis m. xv.
Aquam chloroformi ad ʒi.
ʒi ter die, for a child of one year.

This mixture will sometimes serve throughout the course of the illness, but if the cough becomes loose and bubbling the mixture may be changed to one more stimulating, such as :—

℞ Tincturæ ipecacuanhæ m. v.
Ammonii carbonatis gr. ¼.
Spiritus ammonii aromatici m. v.
Syrupi scillæ m. v.
Aquam camphoræ ad ʒi.
ʒi ter die, for a child of one year.

In young infants cyanosis and restlessness are often increased simply by the abundance of secretion, and then, provided they are otherwise well-developed and strong, it is often a good plan to give an emetic, which will get rid of a great deal of the secretion. Emesis will be obtained by giving half to one teaspoon of ipecacuanha wine. Emetics must not be used in feeble infants, nor when the illness has been prolonged, nor when bronchopneumonia has already developed. During convalescence a good cod-liver oil emulsion should be given, and older children should be sent for a change of air to the country or seaside.

Chronic Bronchitis

Chronic, or recurrent, bronchitis is very common in childhood. Many of the cases are associated with asthma. An exciting cause is often to be found in the unhealthy state of the tonsils and adenoids, infection rapidly passing down to the chest. Chronic bronchitis may also be a legacy from the infectious fevers, particularly measles and whooping-cough, and is often accompanied by some degree of pulmonary fibrosis. A form of hilar bronchitis, in which moist sounds are localised over the roots of the lungs, may accompany tuberculous enlargement of the mediastinal glands.

Symptoms. The symptoms of chronic bronchitis are always more noticeable during the winter months. Cough is the most outstanding symptom, and may be either dry and hard, or loose, depending on the amount of secretion. It is often worse at night, keeping the child awake, or may be particularly troublesome when the child first gets up in the morning. There is likely to be a degree or two of fever, especially in the evenings, and this may continue for weeks at a time, with periodic exacerbations during which the symptoms flare up into an attack of acute bronchitis. Shortness of breath is only complained of in the more severe cases and in older childhood; there is then nearly always a history of asthma, and examination is likely to show some degree of emphysema.

Physical signs are very much as in adults. There is often a poor expansion of the chest. The percussion note is not altered, but the breath sounds are harsh, expiration may be prolonged, and numerous dry and moist sounds are to be heard over both lungs.

The child with chronic bronchitis is generally of spare build, the intercostal spaces being wide and indrawn. Nutrition is interfered with, and the weight is almost always several pounds below the average.

Treatment. A clear upper airway must be provided, and infected tonsils and adenoids should be removed. The operation is, however, no guarantee of cure, and if the sinuses are infected as well the cough will almost certainly continue until they have been dealt with. Other cases are improved by an autogenous vaccine, prepared either from the sputum when that can be obtained, or from a throat swab. Needless to say, a vaccine is of little use unless infected tonsils have first been removed. The diet should be as nourishing as possible, and should include a sufficiency of animal fat, butter, eggs and milk. A teaspoonful of cod-liver oil and malt may with advantage be given thrice daily. The following prescription has often been found serviceable, and may be combined with a sedative cough mixture at night.

℞ Creosoti m. ¼.
Tincturæ benzoini compositæ m. iiss.
Potassii iodidi gr. i¼.
Tincturæ stramonii m. vi.
Aquam chloroformi ad ℥i.

Residence during the winter months at a warm seaside town such as Ventnor or Torquay is very desirable if it can be arranged.

Acute Fibrinous Bronchitis

This rare condition is characterised by the expectoration of fibrinous casts of the bronchi. It has to be distinguished from diphtheritic bronchitis, in which a membranous cast of the bronchial tubes also forms. The chief symptoms of fibrinous bronchitis are cough, urgent dyspnœa, cyanosis, and the expectoration of the casts. During the attack the physical examination resembles an acute attack of asthma, but relief is obtained with the ejection of the cast. The outlook in an attack is good, but relapses are likely to occur.

Treatment is on the same lines as for acute bronchitis. Inhalation of the steam from limewater has been recommended. Potassium iodide in increasing doses is generally held to be the most useful drug internally. Unless diphtheria can be absolutely excluded it would be wise to give a full dose of anti-diphtheritic serum.

Foreign Bodies in the Bronchi

Foreign bodies in the bronchi are met with particularly in young children, owing to their habit of putting things in their mouths. A sudden start or cry may then lead to the inhalation of the foreign substance. All manner of objects may be inhaled in this way, peas, beads, and chips of wood being among the more common. Owing to the right main bronchus being more in the direct line of the trachea, foreign bodies are found almost twice as often in the right as in the left bronchus.

At the time that the foreign body is inhaled there is likely to be a severe fit of coughing or choking, the later history depending on the nature of the foreign substance and its size and shape. Vegetable substances, such as peas or pieces of nut, rapidly decompose and set up an acute bronchitis or broncho-pneumonia within a few days. Mineral substances may remain in the bronchus for some time with surprisingly little in the way of symptoms.

The effects of a foreign body in the bronchus are manifold. Generally within a few days, if not at once, the mucosa at the site of the impaction becomes swollen and shuts off the airway. Bronchial secretions then accumulate behind the foreign body and soon become septic, leading to broncho-pneumonia. Unless the foreign body is removed at this stage, the area of lung beyond is likely to form a large abscess, or may become gangrenous. In other cases the condition gradually passes on to severe fibrosis of the lung and bronchiectasis.

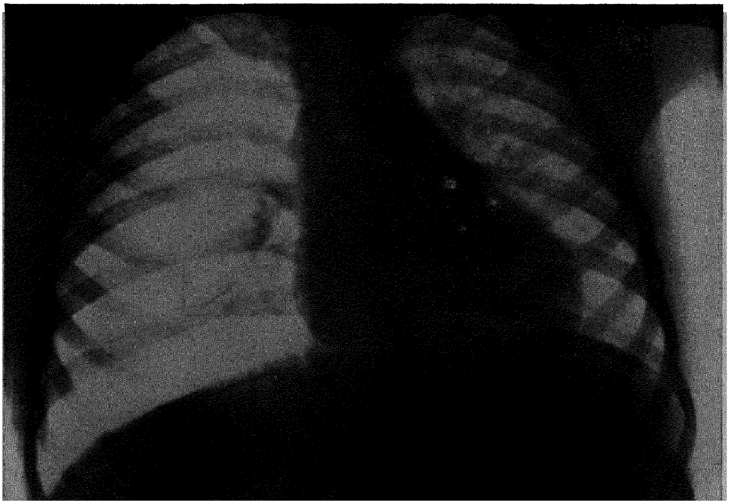

Fig. A. Child aged six years with a piece of brazil nut in the right bronchus. The X-ray has been taken in full inspiration. The right lung is hyperlucent, the right cupola of the diaphragm is flattened and depressed, and the heart and mediastinum are displaced to the left.

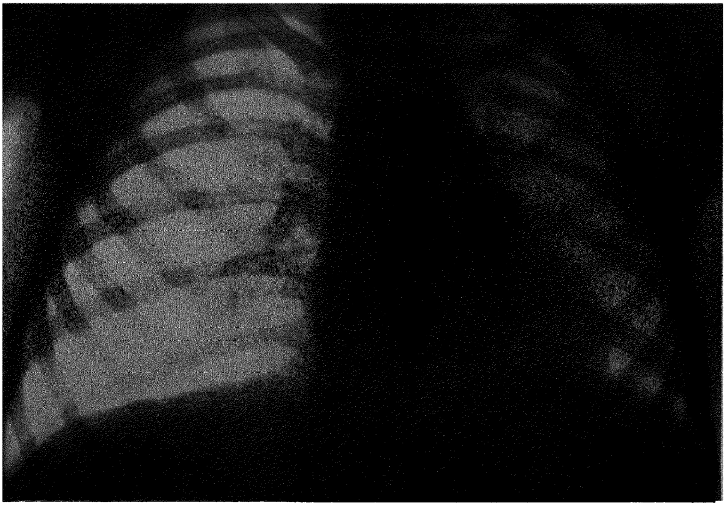

Fig. B. The X-ray is from the same case as Fig. A, but has been taken in full expiration. The right lung remains inflated with air, while the left lung has become opaque from the normal loss of air. The foreign body was successfully removed. With acknowledgments to Mr. V. E. Negus and the *Journal of Laryngology*.

The physical signs vary according to whether air can get past the obstruction. At times the foreign body may act like a valve, allowing ingress of air but preventing egress. Breathing is then likely to be accompanied by stridor, and as the lung beyond the obstruction becomes distended with air the percussion note becomes tympanitic, the breath sounds are much reduced, the heart may be displaced towards the unaffected side, and the diaphragm remains in a position of descent throughout the respiratory cycle. If air can get to and fro past the foreign substance the breath sounds will be tubular, and crepitations with a metallic character may be heard in the lung beyond. When the foreign body completely blocks the airway, as happens eventually in most cases, the air in the lung beyond is soon absorbed, the lung collapses, and with its retained secretions becomes a "drowned lung." The signs then closely resemble an empyema, the percussion note is dull, breath sounds are absent, but the heart may be displaced towards the affected side while the diaphragm remains high both in inspiration and in expiration. When the "drowned lung" becomes infected an abscess forms, the temperature and pulse rise, and the patient rapidly becomes dangerously ill.

Diagnosis. The history of a sudden severe bout of coughing for no apparent reason would be most suggestive. A unilateral character to the signs in the chest also tends to confirm one's suspicions. The signs may vary, and may be those of a unilateral bronchitis, broncho-pneumonia, emphysema or empyema. X-ray examination is essential, and it is invaluable to examine the patient under an X-ray screen. A metallic foreign body will show on the X-ray film. Much help is to be had from X-raying the child during full inspiration and then at full expiration, for in the normal child the alteration in opacity of the lung fields and the different shape of the diaphragm in these two positions is obvious, but when the air cannot escape from a lung owing to a foreign body (obstructive emphysema) the persistence of translucency and of descent of the diaphragm in expiration is diagnostic of the condition even though the object itself may be non-opaque to X-rays (see Plate IV). If doubt remains, expert bronchoscopy should be undertaken. Foreign bodies in the bronchi are generally overlooked because the physician has not entertained the possibility of their presence.

The chance of a foreign body being automatically coughed up is remote. Chevalier Jackson puts it at from 2 to 4 per cent. On the other hand it is generally true that if a foreign body has entered the bronchus an experienced bronchoscopist can usually get it out, but there can be few conditions in which success depends so largely on the skill of the operator. Once the foreign substance is removed, the lung is capable of making a most remarkable recovery, but several months may elapse before complete re-expansion takes place.

ASTHMA

Asthma is a common complaint in childhood, and is a subject which presents many facets, but its complexity can be reduced by dividing the cases into two clinical groups, which differ not merely in their symptoms but also in their etiology and treatment.

The first group consists of what may be called *True Spasmodic Asthma*, the chief features of the attack being a sudden onset, often beginning at night, and in which from the very commencement shortness of breath and wheezing are prominent. These attacks may last for an hour or two, or a day or so, but generally terminate quickly, and although the child may be left with some mild bronchitis for twenty-four hours, as often as not the child is quite well on the day after the attack. In the second form the attack commences with a cough or a cold due to infection in the upper respiratory passages, and after twenty-four hours or so the infection passes down to the chest, producing bronchitis, to which later is added a spasmodic or wheezy element. These attacks, which have resemblances to ordinary bronchitis, may last for several days up to a week, and only gradually pass off. Such attacks may be called *Asthmatic Bronchitis*, to indicate that the chief lesion in the chest is bronchitis, on which asthmatic symptoms are superimposed. The term " Bronchial Asthma " is avoided here, since it is applied to both groups by different observers, and because all asthma is in a sense bronchial inasmuch as it affects the bronchi.

True Spasmodic Asthma

Etiology. Spasmodic asthma occurs more commonly in boys. Heredity exerts a strong influence, for there is a history of asthma or some other allergic condition such as hay fever, eczema, or migraine in the parents or relatives in as many as 70 per cent. of the cases (Bray). The child himself may also have a history of other allergic diseases, the most common example being the child who has eczema as an infant and exchanges this for asthma after a year or so. Urticaria, hay fever, and migraine are also not infrequently associated in the asthmatic child, and the more rare condition of congenital ichthyosis is also likely to be accompanied by asthma.

One of the most important factors in the production of true spasmodic asthma is a state of hypersensitivity or allergy to some foreign substance, contact with which induces an attack. The substance to which the child is sensitive (allergen) needs only to be encountered in minute quantities, and is not injurious to normal children. Occasionally the hypersensitive state is already present at birth, and evidence of it may then appear as soon after birth as the specific substance is encountered, but more often hypersensitivity is acquired after birth by the child coming into intermittent contact with the foreign substance. As a rule the foreign substance is a protein, and it may gain entrance to the body either by being ingested with the diet (ingestants) or by inhalation (inhalants).

Ingestants. These are the proteins of food. The two most common offenders are the lactalbumin of milk and the protein of egg, while the proteins of fish, wheat, and meat come next in order of frequency. Food protein hypersensitivity is particularly likely to be a factor in the asthmas which begin in infancy.[1] It has been shown that food proteins in the mother's diet may be absorbed as whole protein and eliminated in the breast milk, and in this way an infant may be brought into contact with a variety of food proteins at a time when the diet consists only of breast milk.

Inhalants. These are the proteins of dust, particularly the dust from horsehair or feather mattresses and pillows, pollens, and animal emanations, the most important being those from horses, dogs, cats, and rabbits. While ingestant proteins play a prominent part in infancy, the importance of inhalant proteins increases as the child grows older.

A careful history will often give a clue to the particular protein causing a child's attacks, and more precise information can sometimes be obtained by carrying out skin tests to the various common proteins.[2] The tests are performed by scratching the cleansed skin and rubbing in a drop of the protein solution much as in ordinary vaccination; in this way it is possible to test a dozen or more proteins at a sitting. The intradermal injection of proteins is more painful, so that only a smaller number can be done at a time, and the reactions are likely to be more severe. By either method a positive reaction is indicated by the development within ten or fifteen minutes of a branching urticarial wheal at the site of inoculation.

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[1] Donnally, H. *Jour. Allergy*, St. Louis, 1929, 1, 78.

[2] Outfits containing proteins for skin testing are put up by various firms, and they vary considerably in their potency. One of the most reliable sets is prepared by C. L. Bencard Ltd.

Although much is made at the present day of the importance of protein hypersensitivity in spasmodic asthma, other factors are often at work. Thus the condition is undoubtedly more common in nervous, excitable, and highly strung children, and not infrequently an attack is brought on at times of excitement such as children's parties. In other cases attacks are associated with digestive disorders; for instance, simple overfilling of the stomach, particularly at bedtime, may be sufficient to induce an attack. Occasionally attacks of asthma occur with a remarkably regular periodicity, such as once every three or four weeks, so much so as to amount to " cyclical asthma ". These periodic cases are likely to be associated with a faulty metabolism of fat, as shown by ketonuria, and in their management it is necessary to reduce the intake of fat in the diet and to increase the amount of carbohydrate, and it is in these cases that the exhibition of glucose is likely to be beneficial. Another factor of some importance is climate, for there is no doubt that a particular locality will suit some asthmatic children and not others.

Pathology. The changes that take place during an attack consist of spasm of the plain muscle of the bronchi and œdema of the bronchial mucosa, both factors leading to narrowing of the lumen. During the attack there is also likely to be some degree of emphysema, and the blood count sometimes shows a mild eosinophilia. Bray[1] has brought forward evidence of a hypochlorhydria in many of these children, which may at times amount to complete achlorhydria, and good results have been claimed to follow the administration of hydrochloric acid. But it may be pointed out that although a low hydrochloric acid content may make it more easy for whole undigested protein to pass into the intestine, and from there to be absorbed, it does not explain why one child should be upset by the protein of eggs and another by that of milk. Moreover, the low gastric acidity occurs also in children whose symptoms are attributable to inhalant proteins.

Symptoms. Spasmodic asthma often begins in infancy, although at that age the attacks are likely to be mistaken for bronchitis, and their true nature may not be recognised until two or three years later. The attacks in infancy differ, however, from ordinary bronchitis in their sudden onset, their short duration, their quick recovery, and the absence of high fever. After infancy, the attacks resemble those met with in adults. They often begin during the night, the child sitting up in bed, cyanosed, sweating, and making short inspirations and long wheezy expirations. The attack may be over within two or three hours, and the child seem himself again next day, or the spasm may last a day or two, being accompanied towards the end by a good deal of bronchitis.

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[1] Bray, G. W. *Quart. Jour. Med.*, 1931, **24**, 181.

Examination of the chest during an attack shows the lungs in a state of full expansion, the percussion note is hyper-resonant, inspiration is shortened, and expiration is much prolonged and is accompanied by high-pitched sibili and whistling sounds. In addition there is always a considerable degree of emphysema, the area of cardiac dullness may be completely obliterated, and the upper border of the liver dullness may be displaced downwards for one or two spaces. It is important to notice how long the emphysema takes to disappear after the attack is over. As a rule it subsides within a day or two, and this allows of a good prognosis, for if the asthmatic attacks can be overcome the child will be left with a normal chest. On the other hand, when the attacks have recurred frequently over a period of several years the emphysema may persist between attacks, and this affects the prognosis adversely, for even if the asthma can be subdued the child is likely to be left with emphysema and chronic bronchitis.

Treatment. During an attack the child should be allowed to sit up and adopt whatever position is most comfortable. The windows should be opened to admit fresh air. The most successful means of combating an attack is to give a hypodermic injection of adrenalin, 3 minims of a $\frac{1}{1000}$ solution may be given at three years, or 5 minims may be given to older children, and the dose may be repeated in an hour if the attack is not already subsiding. The older methods of burning nitre powders and stramonium leaves should not be used.

The treatment between attacks is directed towards prevention, and success depends upon a thorough attempt to unravel the underlying causes. The history may point to hypersensitivity to some specific protein, and this may be confirmed by skin tests. Commonly the offending article is the dust of the child's bedding, especially when horsehair or feathers are the materials concerned, and these should then be replaced with bedding stuffed with a vegetable fibre such as "kapok". If, on the other hand, the child is sensitive to a food protein it may be possible to exclude the offending article from the diet, or alternatively the child may be desensitised by giving such minute quantities of the specific substance that an attack is not provoked. The amount is cautiously increased each day, always taking care not to precipitate an attack, until finally the child is able to take a normal quantity. Non-specific desensitisation has also been attempted by giving repeated injections of peptone, tuberculin, or bacterial vaccine, but the results are less reliable, and at any rate, in young children, the frequent injections make the treatment irksome.

General measures. Excitement and fatigue are likely to precipitate attacks and should be avoided. Clothing should be light, cellular material next to the skin being most suitable. Excessive wrapping up, or covering the chest with specially prepared wools, are unnecessary and weakening. The diet needs to be easily digestible and nutritious, quite apart from the question of any specific sensitivity. The teatime meal should be a small one, and food at bedtime should be avoided. Mention has already been made of the value of glucose combined with a low fat diet in those cases which show a regular periodicity. A tablespoonful of glucose should be given in water or lemonade at meal times, but it must be understood that the cases for glucose therapy need careful selection.

Drugs. Unless the general and dietetic management of asthma is carefully carried out, medicinal treatment may be rendered largely ineffective, but, with this proviso, much help is to be derived from the prolonged use of suitable drugs. Having tried many mixtures, the writer finds the old prescription of potassium iodide and stramonium as valuable as any. It is as follows :—

℞ Potassii iodidi gr. i¼.
Tincturæ stramonii m. vi.
Tincturæ camphoræ compositæ m. v.
Syrupi simplicis m. xx.
Aquam chloroformi ad ʒi.
ʒi three times a day.

Tolerance of the mixture does not seem to be acquired, and if it is successful in controlling attacks it should be continued until at least six months has elapsed since the last attack. When the asthma is associated with food hypersensitivity a mixture of pepsin and hydrochloric acid may be given at meal times to promote the digestion of proteins. The following mixture for children of two years and upwards is used at The Hospital for Sick Children :—

℞ Pepsini gr. i.
Acidi hydrochlorici diluti m. xv.
Glucosi m. xx.
Syrupi sennæ m. x.
Aquam chloroformi ad ℥i.

Another deservedly popular drug is ephedrine hydrochloride, which is an alkaloid prepared from the Chinese plant Ma-Huang. It is chemically allied to adrenalin but has a more gradual action, and is particularly useful when given as a tablet at bedtime to those children whose attacks come on during the night. If given during the daytime it sometimes has the disadvantage of making the children very sleepy. The dose should be gr. $\frac{1}{4}$ under five years of age, and gr. $\frac{1}{2}$ for older children.

Asthmatic Bronchitis

Etiology. The etiology of asthmatic bronchitis differs in many respects from that of spasmodic asthma. There is seldom a family history of allergic disease, nor is the child himself likely to have suffered from other allergic complaints ; on the other hand, the attacks frequently date from some illness which has left its mark upon the lungs, measles, whooping-cough, influenza, and broncho-pneumonia being the chief offenders, and, in keeping with this, the age at onset is often delayed until a few years after birth. Physical examination is likely to show some degree of pulmonary fibrosis, which may be either slight or extensive, and there is also likely to be evidence of persistent or recurrent sepsis in the throat as shown by unhealthy tonsils, enlarged cervical glands, or nasal catarrh. Attacks of asthmatic bronchitis are more frequent during the cold and wet months of the year, while spasmodic asthma is likely to occur equally all the year round.

Symptoms. A typical attack begins with a cough or cold or running from the nose. After a day or so, infection spreads to the chest and signs of bronchitis appear, to which are added later on the features of asthma. Thus the degree of dyspnœa and cyanosis is greater than would be expected from a simple bronchitis, and both inspiration and expiration are wheezy and accompanied by numerous moist sounds. The attack lasts for several days or even two or three weeks, is accompanied by fever, and clears up gradually as does an ordinary attack of bronchitis. From the readiness with which infection passes down into the chest in these children, it seems likely that the bronchial mucosa is unduly sensitive to the organisms present in the upper respiratory passages. After a time, these children may also develop a sensitivity to the common dusts with which they are in daily contact, such as house dust and the dust of bedding material.

The children are generally thin, below their expected weight, and not infrequently show a badly shaped chest with indrawing of the lower costal spaces and a poor degree of expansion.

Treatment. The treatment during an attack is much the same as for acute bronchitis, with the addition of anti-spasmodic drugs. The child should be nursed in bed. If there is much secretion an emetic dose of ipecacuanha wine will often bring relief. Moistening the atmosphere with steam impregnated with Friar's Balsam has a soothing effect. If there is much nasal catarrh, a gargle of potassium chlorate (gr. 12 to 1 oz.) or glycothymoline should be ordered, or the nose may be sprayed with a solution of menthol gr. 4, eucalyptol m. 4, liq. adrenalin hydrochlor. m. 30, paraffin to 1 oz. For the chest some such mixture as the following will generally bring relief :—

℞ Tincturæ ipecacuanhæ m. v.
Syrupi tolutani m. v.
Tincturæ camphoræ compositæ m. v.
Tincturæ stramonii m. v.
Syrupi simplicis m. xv.
Aquam chloroformi ad ʒi.

Between attacks an attempt must be made to prevent the repeated infections of the upper respiratory passages. A clear airway is important, and if the tonsils and adenoids are unhealthy, they should be removed. It may be pointed out here that while tonsillectomy is useful in a certain number of these children, it is generally entirely without effect in cases of spasmodic asthma. The operation is more likely to be successful when done early, and especially if the lungs have not been damaged by other illnesses. When attacks persist after the operation, help may sometimes be obtained from an autogenous vaccine prepared from the sputum if that can be obtained, otherwise from the flora of the throat. Stock vaccines are less efficacious. It seems likely that the benefit of a vaccine is due as much to its effect in desensitising the child to the common catarrhal organisms, as to raising the titre of immunity against these organisms, and it is therefore important that the vaccine should be commenced in quite small doses, and the dose be only slowly increased.

With regard to drugs between attacks, the potassium iodide and stramonium mixture, already given under spasmodic asthma, is also useful in asthmatic bronchitis. When there is evidence of pulmonary fibrosis, small doses of creosote have often seemed beneficial, and may be ordered in the following prescription :—

℞ Creosoti m. ¼.
Potassii iodidi gr. i.
Extractum malti ad ʒi.

This may be alternated with a course of cod-liver oil and malt.

Lastly, there can be no doubt of the benefit of sea air for these children. When the age of schooling is reached a boarding school at one of the coast resorts is ideal, or if the circumstances do not permit of this, an annual period of convalescence at the seaside should be arranged.

The following table sets out the main differences between spasmodic asthma and asthmatic bronchitis :—

Spasmodic Asthma.	*Asthmatic Bronchitis.*
Attacks of sudden onset.	Attacks of gradual onset, beginning as a cough or cold which goes on to bronchitis, and wheeziness appears after a day or two.
Attacks may last for a few hours up to a day or two, and are apyrexial. Recovery after the attack is often quick, but there may be bronchitic signs for a day or two.	Each attack may last a week or more, is pyrexial, and terminates gradually with residual bronchitis.
Family history of asthma or other allergic manifestations.	Family history negative.
The patient often shows other allergic conditions.	Not associated with other allergic states.
Onset often in infancy.	Onset delayed until three or four years old.
Lungs generally healthy (except for temporary emphysema). X-ray of lungs normal.	Lungs often damaged by previous illness, particularly measles, whooping-cough and broncho-pneumonia. May be X-ray evidence of pulmonary fibrosis.
Skin tests to foods and inhalants often positive.	Skin tests usually negative, although reaction to inhalants sometimes obtained.
Hypochlorhydria frequent.	Gastric analysis normal.
Tonsils generally healthy and tonsillectomy useless.	Tonsils likely to be diseased and tonsillectomy is often beneficial.
Vaccines of no value.	Autogenous vaccines from throat or sputum often useful.

PNEUMONIA

LOBAR PNEUMONIA

Lobar pneumonia occurs at all ages and is quite common in childhood. It has been held that lobar pneumonia is uncommon under two years of age, the pneumonia of infancy being principally broncho-pneumonia. This view has come about largely because lobar pneumonia in young children is a milder disease than broncho-pneumonia and has on the whole a good prognosis, while the fatal cases are nearly always of broncho-pneumonic type. Actually, of 50 consecutive cases of pneumonia under two years of age seen by the author, one-third were due to lobar pneumonia, and McNeil [1] and others in a detailed study reckoned that 45 per cent. of cases of lobar pneumonia in children under twelve occurred during the first two years. In their series of 534 cases up to twelve years of age 27 per cent. were broncho-pneumonic and 73 per cent. were due to lobar pneumonia.

The pathology of lobar pneumonia in children does not differ from that of adults, but a greater variety of clinical types is met with, and complications are also more numerous.

Symptoms. As a rule the onset is sudden, although there is sometimes a history of coughing for a day or two previously. Vomiting at the onset is so usual that during the early stages it may be of real help in suggesting the diagnosis. The two conditions in children which characteristically have a high temperature and vomiting at their beginning are lobar pneumonia and follicular tonsillitis. In lobar pneumonia the temperature mounts rapidly to 103° or 104° F. and the child becomes hot and drowsy, in fact drowsiness at the onset is usually obvious enough to be remarked by the parents. It may persist, or may give place to restlessness or delirium as the disease advances, especially if the infection is situated in an upper lobe. Occasionally the onset is attended by a shivering fit, or in young children there may be one or more convulsions. Fits at this stage have no prognostic significance, it is only when they occur later in the disease that they may be of serious omen, indicating perhaps some such complication as meningitis or intracranial thrombosis. As a rule the bowels are constipated from the onset, although in babies an initial diarrhœa may occur.

As the temperature rises the pulse and respirations increase in rate, the latter out of proportion to the former, so that the normal 4 to 1 ratio between the pulse and respiration may drop to a

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[1] McNeil, MacGregor and Alexander. *Arch. Dis. Child.*, 1929, 4, 84.

3 to 1 or even a 2 to 1 ratio. The respirations may run up to sixty or even eighty per minute. Older children may complain of pain in the chest, due to accompanying pleurisy, and if the pleura overlying the diaphragm is involved the pain may be referred to the top of the shoulder. On the other hand pain from the lower lobes may be referred to the abdomen, and if this should be on the right side the condition may simulate appendicitis. The presence of pain in younger children must be gauged by their restlessness, the character of their cry, and the shallowness of their respirations.

Once the disease is established the appearance of the child is characteristic, as he lies with flushed cheeks and bright eyes. The skin is hot and dry. Herpes may occur round the mouth just as in adults, and is usually situated on the same side as the affected lung. Coughing is slight, or may be suppressed. The respirations are short and rapid, and may show an inverted rhythm—inspiration and expiration being linked together so that the pause which ordinarily occurs at the end of expiration takes place at the end of inspiration—and not infrequently a slight grunt can be heard at the beginning of expiration. The alæ nasi may dilate with each breath. In childhood there is no sputum, or what sputum there is is swallowed.

The temperature in a typical case is sustained at 103° to 104° F. until the crisis, which occurs between the third and ninth day, most commonly about the fifth day. In young children the temperature may show a daily swing of two or three degrees. Another not uncommon happening is for the temperature to drop suddenly several degrees, and in a few hours to be up again to its previous height. Such a pseudo-crisis is likely to occur about twenty-four hours before the true crisis.

Physical examination in the early stages is often negative. The earliest signs consist of diminished movement on the affected side, and diminished air entry over the affected lobe. Later the percussion note becomes dull, without reaching the stony dullness of fluid, and tubular breath sounds are audible over the dull area. As a rule crepitations do not appear until the crisis approaches, and are then fine and crackling, but in infants it is not uncommon for a few coarser crepitations to be heard in other parts of the chest throughout the course of the disease. In some cases both tubular breathing and crepitations may be inaudible unless the child can be encouraged to take one or two deep breaths. It must also be pointed out that lobar pneumonia does not always affect an entire lobe, and unless the chest is thoroughly examined a small area of consolidation may easily be overlooked, particularly when situated high up in the axilla or at the apex of an upper lobe. Another peculiar feature is the rapidity with which the physical signs may develop, so that within a few hours of searching the chest for pneumonia and finding nothing, a second examination may show an impaired note and loud tubular breathing.

The examination of the blood shows a polymorph leucocytosis. The white cells may run up to 30,000 per c.mm., of which 70 to 80 per cent. are polymorphs. Albuminuria is constantly present.

Following the crisis, resolution of the lung is rapid, and is complete within a week. In the few cases where the temperature falls by lysis, resolution of the lung may be delayed over a few weeks, but the condition is very unlikely to go on to pulmonary fibrosis.

Varieties. These are more frequent in children than in adults.

Abortive. After a typical onset the temperature falls on or before the third day, and signs of consolidation of the lung, if they appear at all, are evanescent.

Apical. Pneumonia occurring at the apex, more often on the right side, may be looked upon as a special variety because it is so likely to be accompanied by nervous symptoms, of which restlessness and delirium are the most troublesome. Signs of meningeal irritation, such as stiffness of the neck muscles or actual head retraction, and Kernig's sign may also occur, and may easily lead to confusion with meningitis.

Central. In this form the consolidation is situated near the hilum of the lung, and is surrounded by normal lung tissue. The pleura often escapes, and the respiration rate may then be scarcely raised. Examination of the chest may fail to reveal any signs of consolidation, and therefore the diagnosis is likely to present great difficulty, for one has to rely largely on the nature of the onset and the general appearance of the child, and it is not until the fever has terminated by crisis that one feels sure of one's ground. There are also cases in which the signs of consolidation do not appear until within a few hours of the crisis, or may even appear up to twenty-four hours after the crisis. This is probably accounted for by a spreading form of consolidation which begins centrally and slowly creeps towards the surface.

The following case illustrates the difficulties that this type of case presents. A little girl aged three years was in good health until August 8th, when she vomited and her temperature rose to 104° F. The temperature remained at that level until August 14th when it fell to subnormal, only to rise again within a few hours to 105° F. Examination at this stage showed no physical signs except that the child was very flushed. A diagnosis of central pneumonia was made, the falling temperature being regarded as a pseudo-crisis. Twenty-four hours later the temperature fell to normal, and recovery was uneventful.

Pneumonia Migrans. In this condition one lobe becomes consolidated after another. There may be a crisis with an interval of a day or so before the attack spreads to the next lobe, or the disease may travel more rapidly without a fall in temperature until such time as the whole of both lungs has been involved. The crisis may thus be delayed for three or four weeks. As a rule resolution takes place rapidly in the lobes first affected, so that they are able to support life by the time the consolidation has passed on to other areas.

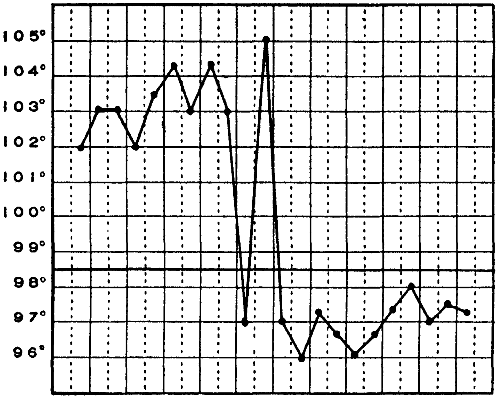

FIG. 51. Temperature chart of a child aged three years, with central pneumonia. The clinical history is given in the text.

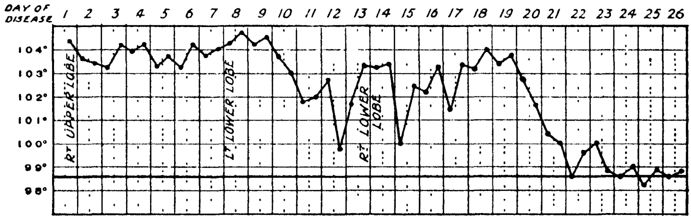

FIG. 52. Temperature chart of a boy aged two and a half years, with *pneumonia migrans.*

Complications. The various complications of lobar pneumonia are more likely to be met with during early childhood than in later years. The most important complication is empyema, which occurs in roughly 14 per cent. of the cases. Occasionally pus begins to form in the pleura before the pneumonic process has reached its crisis (syn-pneumonic empyema), but more usually the signs of empyema are delayed until from three to seven days after the crisis (post-pneumonic empyema). Acute otitis media is another not uncommon complication, and in young children may raise suspicions of pneumococcal meningitis by giving rise to some stiffness of the neck. Suppurative pericarditis is a complication which is principally met with in quite young children, and even then is, fortunately, rare. Beyond giving rise to temporary pericardial frictions there are seldom other distinctive signs, and the condition is therefore easily overlooked. Meningitis, suppurative arthritis, and peritonitis, must also be mentioned, but are uncommon. Acute nephritis is an occasional complication ; it may be met with during the height of the fever, and is generally of short duration.

Diagnosis. An illness of sudden onset with vomiting, drowsiness, cough, and high fever as the initial symptoms, must always suggest lobar pneumonia. Tonsillitis may begin in much the same way, but the routine examination of the throat will prevent error. Acute otitis media and acute pyelitis must also be excluded. When the pleura over the right lower lobe is affected the pain may be referred towards the right iliac fossa, and the muscles of the abdominal wall may also be so tense that there is real difficulty in deciding whether the child has pneumonia or appendicitis. The temperature is usually higher in pneumonia, and the disproportion between the respiratory and pulse rates is a valuable guide. In a doubtful case it is wise to delay a diagnosis for twelve hours or so, by which time further evidence of pneumonia, such as impaired movement of the chest and a deficient air entry, should have appeared. When the upper lobe of the lung is affected the delirium and stiffness of the neck may be most suggestive of meningitis, and it may even be necessary to resort to a diagnostic lumbar puncture. X-ray examination of the chest may also prove helpful (see Plate V). The distinction between lobar and broncho-pneumonia is deferred until the next section.

Prognosis. Barring complications, the outlook for the child with lobar pneumonia is good. In 386 instances in children under twelve years of age McNeil found the mortality to be 7 per cent. The mortality is highest in the early years. The crisis is a much less weakening event in children, and within a few hours the child may be sitting up and ready to play with his toys. One attack of lobar pneumonia does not confer immunity, and a child may have as many as three or four separate attacks.

Treatment. The child should be nursed in a warm room at a temperature of about 62° F. Fresh air is essential, and should be obtained from an open window, but in foggy or damp weather a coal fire and an open door will serve the same purpose. Draughts must be excluded by the use of screens. A steam tent is likely to embarrass the breathing and will do more harm than good. The clothing should be warm and light, and a gamgee pneumonia jacket does very well; it should be made to do up with tapes down the sides so that the chest may be easily and quickly uncovered for examination. The diet needs to be mainly of fluids such as milk, bread and milk, fruit drinks, and broths. There is no need to limit the amount of fluid, and during the height of the fever the child's thirst should be satisfied.

Drugs are chiefly of use in relieving symptoms. If coughing is frequent, as it may be in the early stages, small doses of opium are of value, but opium should only be given during the first day or two. The following prescription will serve for a child of two years :—

℞ Tincturæ camphoræ compositæ m. v.
Syrupi scillæ m. v.
Spiriti ætheris nitrosi, m. v.
Syrupi simplicis m. x.
Aquam ad ʒi. ʒi ter die.

When at the onset the respirations are shallow and rapid, and the child is restless because of pain, much relief may be given by a linseed poultice. The addition of a teaspoonful of mustard to the linseed increases the counter-irritant effect of the poultice. Warm antiphlogistine spread on lint makes a soothing application, but has the disadvantage of hindering the examination of the chest, and is too heavy for young children.

Hyperpyrexia may call for treatment. When the temperature rises above 104·5° F. the child should be given a tepid sponge to bring the temperature down by one or two degrees.

Adequate sleep is essential, and a tepid sponging is one of the best means of promoting sleep. If this fails, a small dose of chloral (1 gr. per year) may be given at night-time. As the crisis approaches, a watch should be kept for signs of cardiac failure, such as increasing cyanosis and dyspnœa, a rising pulse, or dilatation of the heart. Small doses of tincture of digitalis (m. 2½ ter die to a child of five years) are then valuable, and also at this stage brandy may be given. It needs to be diluted with water, and should be given between meals. Half a teaspoonful should be allowed for a child of two years. Oxygen is also of value, and may quickly relieve the cyanosis. Although an oxygen tent is the most effective means of raising the intra-pulmonary oxygen concentration, it is not as a rule required for lobar pneumonia in children, and is more fully considered under broncho-pneumonia. An alternative method is to give the oxygen through a small nasal catheter if the child will tolerate it, the oxygen being bubbled through warm water ; it should be given for ten minutes in each hour. The catheter should be attached to the cheek by strapping so as to keep it in position even if the child moves his head. As a second alternative oxygen can be given through an open mask held close to the face,but this is much less efficacious. If giving oxygen causes the child to struggle, any benefit from it will be lost, and it should be discontinued. Cardiac stimulants, such as camphor gr. ¼ in oil hypodermically to a child of three years, or strychnine gr. $\frac{1}{200}$th, should be at hand in case of an emergency.

Sulphanilamide is ineffective in this disease, but the use of sulphapyridine calls for comment since it exerts a bacteriostatic effect on pneumococci. If it is to be used, full doses must be employed, otherwise the fever may either not settle, or may recur when the drug is stopped, and administration should go on for forty-eight hours after the temperature has fallen. While sulphapyridine will abort the fever in many cases, and lessen the chance of complications, the child is apt to be left in a very low and spiritless condition ; bearing in mind the favourable outlook of this disease in children, the drug may be reserved for those cases in which the illness is violent as judged by the degree of toxæmia, or if the course is prolonged, more than one lobe is involved, or there is an accompanying pleurisy. The diagnosis from broncho-pneumonia must also be in no doubt, for in the latter condition there should be no delay in giving sulphapyridine.

Recently much benefit has been reported in adults from the use of Felton's anti-pneumococcal serum. The good outlook in childhood renders the use of this serum unnecessary, while the preliminary typing of the infecting organism is difficult to do in children owing to the absence of sputum.

BRONCHO-PNEUMONIA

Etiology. In childhood broncho-pneumonia is very much more common during the first two years of life than later on, and in the early years carries a high mortality, in fact deaths from pneumonia in infancy are generally due to broncho-pneumonia. Upper respiratory infections commonly precede broncho-pneumonia, and so, as one would expect, the disease is more common during the winter. Broncho-pneumonia differs from the lobar variety in its tendency to pick out weak children and those debilitated by such lowering diseases as diarrhœa and marasmus, in which broncho-pneumonia is often a terminal event. In these feeble infants broncho-pneumonia may develop without provoking any obvious reaction, the temperature remains subnormal, and the presence of consolidation in the lung is often not discovered until post-mortem examination. Of the infectious fevers there are two that are particularly likely to be complicated by broncho-pneumonia, namely measles and whooping-cough, and they owe their mortality largely to this complication. Rickets also renders a child more liable to broncho-pneumonia, and the presence of the former makes the prognosis grave.

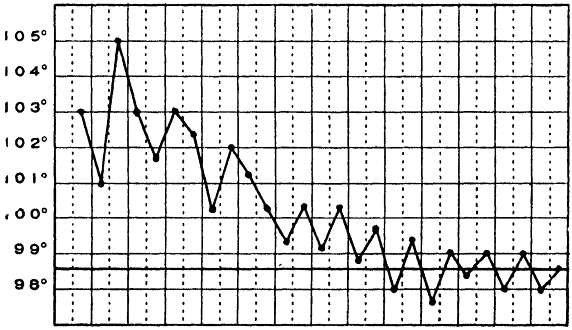

FIG. 53. Temperature chart of a boy aged eighteen months, with broncho-pneumonia.

Pathology. The pathology of broncho-pneumonia is the same in children as adults, and consists of an acute interstitial inflammation of the lung. At autopsy the surface of the lung shows a patchy appearance, made up of slate-coloured areas of collapse, with others purple or greyish-yellow with consolidation, while here and there may be small patches of emphysema. The areas of consolidation are often too small to be recognised individually during clinical examination, but they may become large enough to coalesce and give a confluent broncho-pneumonia, in which case the whole of a lobe may present the signs of consolidation. The most common causal organisms are streptococci and pneumococci.

Symptoms. The onset is sometimes swift, with high temperature, rapid respirations, cyanosis, and occasionally convulsions, but there is generally a preceding history of cough for two or three days, and the signs of bronchitis. Once the disease is established the clinical picture is one of severe illness with much respiratory embarrassment, more so than in the case of lobar pneumonia. The child is most comfortable if propped up on pillows. The expression is anxious, the eyes are dull and listless, the tongue is dry, and the complexion is pallid. Resentful of examination, the child is often too weak to cry, and merely tries to push away the stethoscope. Slight alterations of position are likely to bring on a fit of coughing, which leaves the child exhausted. Respirations are rapid, and may run up to 100 per minute, and the respiratory cycle becomes inverted, that is to say, a pause occurs at the end of inspiration instead of at the end of expiration. Respiration is likely to be accompanied by a soft grunting, and there is much dilatation of the alæ nasi. The cough is variable, but is usually short and frequent, although in young infants it may be entirely absent, and there is no expectoration. The temperature fluctuates daily between 100° and 103°, and eventually terminates by lysis, or may show fresh rises as new areas of lung become involved. Remissions and relapses are not uncommon, and may carry the disease over a period of three or four weeks before the temperature finally settles down. The pulse rate is relatively higher in broncho-pneumonia than in lobar pneumonia, and in young infants may become almost uncountable as signs of exhaustion appear.

On examination there is likely to be some retraction of the lower intercostal spaces, particularly during infancy when the chest wall is soft and yielding. The percussion note varies ; it may be unchanged or there may even be some hyper-resonance owing to compensatory emphysema. As the areas of consolidation become more extensive, a patchy impairment of the note can be detected, but downright dullness is rarely found. The greatest value attaches to auscultation. Areas of harsh breathing may be heard, particularly near the bases of the lungs, and fine crackling crepitations are audible during inspiration. The crackling quality of these moist sounds denotes that they are produced in areas of consolidation, and they are typical of broncho-pneumonia, but there may also be much bronchitis with so many coarse râles that the finer crackles are almost obscured. Tubular breath sounds can often be brought out by getting the child to breathe deeply. Towards the end of an exhausting broncho-pneumonia in infants, the respiratory and cardiac rhythm may become very irregular. The breathing may show a Cheyne-Stokes rhythm, or there may be periods of actual apnœa in which the baby becomes leaden-coloured, the heart slows, and the pulse becomes imperceptible. After a few gasping respirations the infant may temporarily recover, but at this stage the outlook is very grave, and death usually follows within a few hours.

X-ray examination shows an opaque mottling over the consolidated areas (see Plate V).

Complications. Empyema is not so often a complication of broncho-pneumonia as of lobar pneumonia, although most cases of streptococcal empyema are secondary to broncho-pneumonia. Suppuration of the lung may occur, and may even proceed to gangrene. Diarrhœa may be a troublesome complication, especially in infants, and may prove rapidly exhausting, or it may be so prominent a symptom at the onset as to mislead one into regarding the bowel disturbance as the primary illness. Otitis media, pericarditis, or meningitis may occur, while in enfeebled children venous thrombosis may be a terminal event. The superior longitudinal sinus, or one or both renal veins, are likely situations. A variable degree of fibrosis is a frequent sequel.

Diagnosis. In a child who has the appearance of severe illness, coupled with fever, rapid grunting respirations, and fine crackling crepitations over one or both lungs, the diagnosis of broncho-pneumonia is an easy matter. There may be the greatest difficulty in infancy in distinguishing between bronchitis and broncho-pneumonia, but generally speaking, in the latter the child appears more gravely ill, and the respirations and pulse are more rapid than a simple bronchitis would account for. The diagnosis from lobar pneumonia should not, at any rate in older children, present much difficulty, and provided that one is prepared to recognise the frequency of lobar pneumonia in infants, even at that age the distinction between the two forms of pneumonia can be made with confidence. An attack of pneumonia which comes on suddenly, beginning with vomiting or a fit, affecting an infant previously in good health, and running a course of three to seven days, the temperature falling by crisis, is characteristic of the lobar variety. On the other hand a pneumonia which is preceded by upper respiratory tract catarrh, affecting a weakly child or one already enfeebled by other illness, and running a severe course with much prostration and eventually settling by lysis after perhaps one or more remissions, is characteristic of broncho-pneumonia.

Doubt will sometimes arise whether a lung is broncho-pneumonic or tuberculous. A history of recent exposure to tuberculous infection is a most important point. Tuberculin skin tests are likely to be deceptive, as they may be negative in so severe an illness as tuberculous broncho-pneumonia. The presence of tuberculosis elsewhere, such as enlarged mesenteric glands or miliary tubercles on the choroid, should be looked for. Lastly, it must be remembered that both measles and whooping-cough may begin with broncho-pneumonia, and that the typical features of these infectious fevers may then be lacking. If there is any reason for suspecting that the broncho-pneumonia is masking an infectious illness the child should be isolated.

Prognosis. Broncho-pneumonia is always a serious illness, particularly so during the early years. Thus of 41 cases under three years of age the mortality was 56 per cent. Of those under one year 73 per cent. died, in the second year the mortality fell to 47 per cent., and in the third year to 20 per cent. Coincident diseases such as rickets, congenital heart disease, or the infectious fevers, affect the prognosis adversely. Complete recovery may take several weeks or months, and there is a great likelihood of some degree of permanent fibrosis, which in the more extensive cases may go on to bronchiectasis.

Treatment. Broncho-pneumonia is so likely to be a long and exhausting illness that from the outset the child's strength must be husbanded as much as possible. Skilled nursing is absolutely essential. The general treatment is on similar lines to that of lobar pneumonia. Fresh air is invaluable, although the child must be protected from draughts. In the warmer months it is a great advantage if the child can be nursed on a balcony in the open air, and this is particularly the case when the illness drags on for two or three weeks. The use of a steam tent has rightly gone out of fashion in this disease. The effect of the moist atmosphere was to increase the secretions in the chest, and so add to the respiratory embarrassment and waste the child's strength. There are no circumstances that permit of the use of a steam tent in broncho-pneumonia. As a rule poultices and plasters to the chest should be avoided, as owing to their weight they are likely to increase the infant's difficulties in breathing.

The diet must be easily digestible, but in an illness which is likely to be prolonged it must be nourishing. During infancy, breast milk, peptonised milk, or a half cream dried milk will be suitable. After infancy, broths, egg custards, milk puddings and thickened milk foods should be given, and there is no need to restrict the intake of fluid.

The value of the sulphanilamide group of drugs is undoubted, and they should be started as soon as the diagnosis has been made. The table of dosage set out on p. 715 should be followed, for unless enough is given, not only are the results likely to be unsatisfactory, but the organisms may rapidly become drug-resistant. Owing to the different organisms that may be concerned, sulphapyridine is as a rule the most suitable preparation. The benefit is as apparent when broncho-pneumonia complicates measles or whooping cough as it is in ordinary broncho-pneumonia, but when the illness accompanies influenza, the effect of sulphanilamide treatment is at times disappointing.

Except for the sulphanilamides, drugs will only be employed to relieve symptoms. When coughing is troublesome and there is much secretion, a stimulating cough mixture such as the following will be found useful :—

℞ Tincturæ ipecacuanhæ m. v.
Tincturæ belladonnæ m. iii.
Ammonii carbonatis gr. ¼.
Spiriti ammonii aromatici m. iii.
Glycerini m. x.
Aquam ad ʒi.

Dose :—ʒi three or four times a day, for a child of a year.

As a general rule sedative cough mixtures are contra-indicated, and morphia should be avoided. Emetics are seldom employed at the present time, as the type of case which is bad enough to need an emetic is too ill to stand the strain of emesis. The use of tepid sponging should be remembered, not only if there is hyperpyrexia, but also as a means of inducing sleep.

A careful watch needs to be kept on the heart for such signs of incipient cardiac failure as a rising pulse, softening of the first sound, increasing cyanosis, or enlargement of the liver. The addition of 2 or 3 minims of tinct. digitalis to the cough mixture may be made with advantage in thwarting these symptoms. When cardiac failure has appeared, the withdrawal of an ounce or two of blood from a vein is a most useful measure, and one that is probably not used often enough.

Oxygen is certainly of value provided that it is not kept in reserve until the child is moribund. It is most effectively given in an oxygen tent,[1] which seems particularly suited to infants and young children, who, while often resenting the older methods, appear quite at home in the tent. Expert nursing is of course necessary. The oxygen concentration should be checked every two hours, and should be maintained at 40 to 45 per cent. until obvious improvement sets in. The concentration may then be gradually reduced and the child be allowed out of the tent for short periods ; about forty-eight hours later the tent may be discontinued. During the height of the illness clinical examinations should be reduced to the barest minimum, as the disturbance entailed and the sudden drop in oxygen concentration may quickly turn the scale against recovery. As an alternative method, oxygen may be given through a nasal catheter, after bubbling it through warm water or alcohol, and should be given for ten minutes in each hour. An easier but much less efficient method is to give the oxygen through a funnel held close to the child's face. Neither of the latter methods should be used if they make the child resentful or restless.

In severe cases, especially during infancy, attacks of collapse may occur, calling for stimulant treatment. One of the best methods is to put the infant into a warm mustard bath for five minutes (1 oz. of mustard to 1 gallon of water at a temperature of 100° F.), wrapping the infant afterwards in a warm blanket. Brandy is also a useful restorative ; 10 drops may be given three-hourly up to three months, and 20 drops at six months. When the respirations become irregular and shallow, due to the failure of the respiratory centre, the inhalation of oxygen (95 parts), and carbon dioxide (5 parts), should be used in the place of pure oxygen. Hypodermic injections of camphor (gr. $\frac{1}{4}$ in oil), or of strychnine (gr. $\frac{1}{200}$th), for a child of one year, will serve as temporary stimulants.

In contrast to lobar pneumonia, serum treatment is worth a trial in broncho-pneumonia because so many of the cases are streptococcal. An intra-muscular injection of 3 c.c. of concentrated polyvalent antistreptococcal serum may be given, and if there is an improvement the dose may be repeated in twenty-four hours.

The treatment of broncho-pneumonia is not complete until every effort has been made to ensure a proper healing of the lung. Open air treatment on a balcony and breathing exercises during convalescence are helpful, but a change of air to the country or seaside is the best means of restoring the lung to a healthy state.

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[1] The portable tents supplied by Oxygenaire Ltd. have proved very efficacious

PULMONARY FIBROSIS AND BRONCHIECTASIS

Bronchiectasis and pulmonary fibrosis are closely related to one another, both from a pathological and clinical point of view. Pathologically there is invariably some degree of fibrosis of the lung accompanying all cases of bronchiectasis, and clinically it is often impossible to make any distinction between the two. The recent use of lipiodol in the investigation of cases of pulmonary fibrosis has served to emphasise how frequently bronchiectasis coexists.

The most common forerunner of pulmonary fibrosis is bronchopneumonia, the interstitial inflammation of the lung becoming converted into young fibrous tissue which eventually leads to permanent scarring. At the same time the inflammation of the bronchial walls, which is an essential feature of bronchopneumonia, causes the walls to weaken and allows of their distortion by the contracting fibrous tissue in the neighbouring lung. Some degree of bronchiectasis may also follow chronic bronchitis, or may be the sequel to an empyema which has either escaped recognition or has been imperfectly drained.

Symptoms. The symptoms of pulmonary fibrosis and bronchiectasis in children closely resemble those in adults. Cough is nearly always present, and may at times be so spasmodic as to suggest whooping-cough. The amount of sputum varies considerably, not only in different children, but at different times in any one child. An absence of sputum may be due to the bronchiectatic cavities being dry, or to the sputum being swallowed. The actual coughing of sputum is most likely to happen in the early morning when the child first gets up, or at bedtime. When the cavities are secondarily infected the sputum may be highly offensive and taint the breath, but in many cases the sputum is odourless. It is not infrequently tinged with blood. Shortness of breath on exertion is a common complaint. The appetite is usually poor, due no doubt in part to the constant swallowing of sputum, and if the condition is at all severe growth is interfered with, and the child gradually becomes stunted in height and much below the average weight. The temperature is likely to hover around 99° F., with an occasional burst of high fever lasting for a few days and associated with an increased catarrh of the affected area of lung. In the more severe cases there is a considerable degree of cyanosis, together with clubbing of the fingers and toes.

The signs over the chest vary considerably, in fact in mild cases it may be almost impossible to detect anything wrong, but as a rule the affected side of the chest is flattened and expansion is diminished, the apex beat is likely to be drawn towards the affected side, and the trachea may be similarly displaced. The percussion note and the quality of the breath sounds depend very much on whether the lung is dry at the time of examination, or whether the dilated bronchi and bronchioles contain secretion. When the lung is dry the percussion note is resonant or may even be hyper-resonant, and breath sounds will be either tubular or amphoric according to the degree of bronchial dilatation. More commonly there is diminished resonance, and the breath sounds are accompanied by coarse metallic moist sounds. Although any area of the lungs may be involved, the commonest site is at the bases, and the left more frequently than the right. There is often some general bronchitis as well, with moist sounds scattered over both lungs. Occasionally when there is much thickening of the pleura, or the bronchial dilatations are filled with secretion, the note on percussion may be dull and the breath sounds so reduced as to suggest the presence of an empyema, but the long history and the displacement of the mediastinum towards the affected side with the presence of moist sounds on deep breathing are points which distinguish a fibroid lung from an empyema.

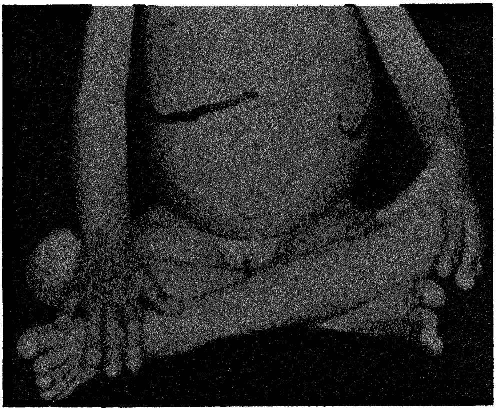

FIG. 54. Girl aged five years with gross clubbing of the fingers and toes, and amyloid enlargement of the liver and spleen, the result of chronic empyema and severe pulmonary fibrosis.

X-ray examination generally shows heavy shadows over the affected area, and if the diaphragm is tethered by adhesions to

PLATE V.

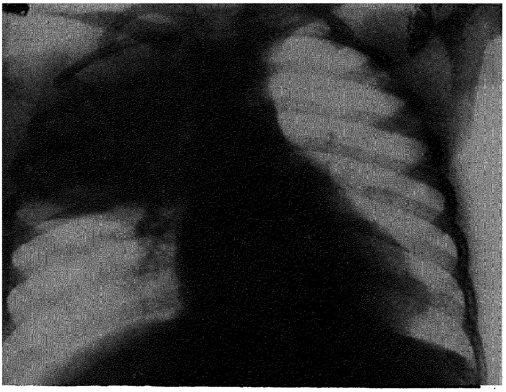

FIG. A. Lobar pneumonia of right upper lobe in boy aged seven years.

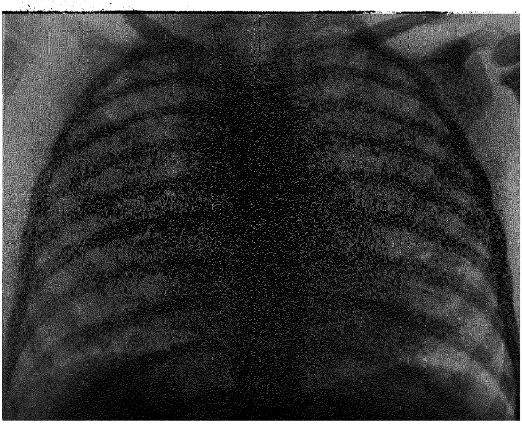

FIG. B. X-ray of a girl aged two and a half years, showing diffuse broncho-pneumonia over both lungs —in her case, a complication of measles.

[*To face p.* 364.

PLATE VI.

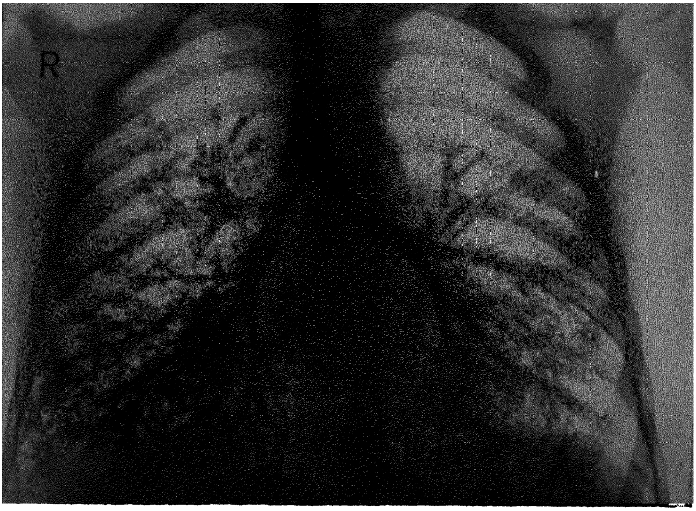

FIG. A. Normal. To show the appearance of lipiodol in a normal chest. Child aged four and a half years. (By courtesy of Dr. Bertram Shires.)

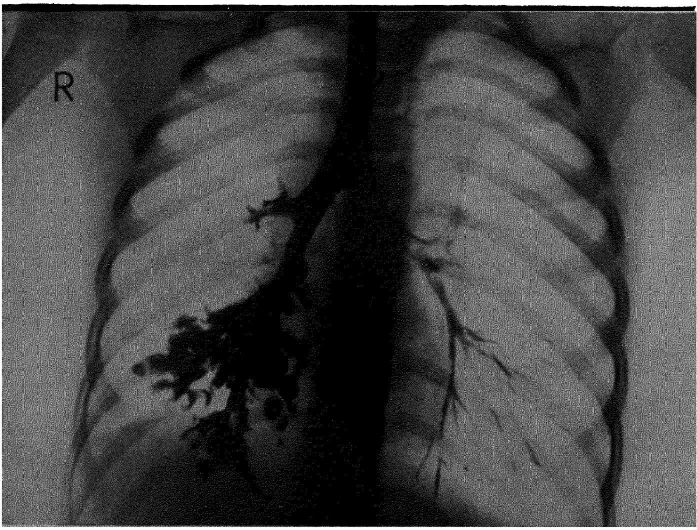

FIG. B. Bronchiectasis with lipiodol. To show the appearance of lipiodol in bronchiectasis of the right lower lobe. Child aged four and a half years. (By courtesy of Dr. Bertram Shires.)

[*To face p.* 365.

the lung its outline will be irregular. Examination under the X-ray screen reveals the diminished movement of the affected side, while any irregularities of the diaphragm can be seen to increase during deep inspiration. It sometimes happens that the affected area of lung is behind the heart shadow, and the X-ray film may then show a triangular opacity with its apex near the root of the left lung and the base reaching down to the left dome of the diaphragm. This shadow is caused by collapse of the left lower lobe, and may be due in the first place to broncho-pneumonia or possibly a foreign body. Unless the collapsed area re-expands, it is likely to go on to fibrosis and bronchiectasis—a condition which has been called " atelectatic bronchiectasis." The right lower lobe may be similarly affected.

Diagnosis. A history of chronic cough, dating perhaps from a severe attack of pneumonia, with cyanosis, finger clubbing, and stunting of growth, together with displacement of the mediastinum towards the affected lung, will suggest the right diagnosis. Pulmonary tuberculosis is not likely to cause confusion, as in childhood the fibroid form of phthisis is very uncommon. X-ray examination is often of assistance, and in doubtful cases an intratracheal injection of lipiodol may be used to outline the bronchial tree. In children the introduction of lipiodol should be carried out under general anæsthesia. A skilful lipiodol X-ray gives precise knowledge of the extent and localisation of the bronchiectatic area (see Plate VI), and this is not only necessary when surgical treatment is contemplated, but also if medical treatment by means of postural drainage is to be successful.

Prognosis. Pulmonary fibrosis and bronchiectasis runs a long course, usually lasting for several years. The course is likely to be punctuated by attacks of re-infection in the affected area, accompanied by high fever and fresh signs of consolidation. If the child is seen for the first time in one of these attacks a diagnosis of pneumonia is very likely to be made, but as a rule the temperature settles quickly, and as the pneumonic signs quickly slip back into those of fibrosis the original scarring comes to be recognised.

It is disputed whether bronchiectasis in childhood is capable of recovery. Findlay and Graham [1] followed up 32 children for six years, and found that during this period 12 had died after an illness which averaged two and a half years, while in 8 others the condition had grown worse. On the other hand, Nobecourt [2] considers that recovery from bronchiectasis not infrequently

[1] Findlay, L., and Graham, S. *Arch. Dis. Child.*, 1931, **6**, 1.

[2] Nobecourt, P. *Précis de Med. des Enfants*, Paris, 1926, 392.

takes place. It is likely that the milder degrees of pulmonary fibrosis with some slight bronchial dilatation may clear up after some years, but when once the classical picture of bronchiectasis has developed the condition should be regarded as grave, and likely to progress slowly to a fatal termination. Death may be the result of chronic toxæmia, broncho-pneumonia, or intracranial abscess.

Treatment. Prevention is all important in a disease which defies treatment, and with this object in view great attention must be paid to the thorough convalescence of children after broncho-pneumonia. When fibrosis of the lungs has appeared, the general health must be maintained at as high a standard as possible. Fresh air, preferably at the seaside, daily exercises not pushed to the point of fatigue, and a good nourishing diet are essential. Preparations of cod liver oil and malt are useful. When the cough is very moist, or if there is actually a daily expectoration of sputum, treatment by posture should be tried. The practice of inverting the child over the side of the cot morning and evening, and, while in this position, encouraging him to cough and so clear his lungs, is sound in principle but is hardly sufficient. The position in bed should be such that the damaged area of lung can drain adequately during the night, and at first the child should also rest in this position for two hours each

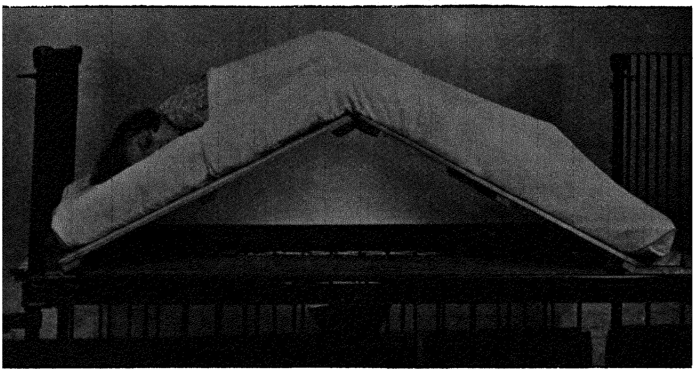

FIG. 55. Postural drainage of the posterior portions of the lower lobes in bronchiectasis. The bed is constructed by using a wooden mattress hinged across the middle. The centre of the bed can then be raised to the desired height by inserting blocks of varying width at the end of the mattress. In the illustration the centre of the bed is raised 15 inches.

day. Fig. 55 shows how a simple " bronchiectasis bed " can be constructed, to drain the posterior portions of the lower lobes, which are the most commonly affected areas. The right middle lobe, and the anterior portions of the lower lobes are drained by the child lying on his side with the affected lung uppermost and the foot of the bed raised 15 inches. The upper lobes are of course drained in the upright position. In a successful case the amount of sputum quickly falls, the temperature settles, and the appetite and general condition improve. If the sputum is offensive a Burney Yeo mask should be worn during part of the daytime, 4 or 5 drops of the following mixture being applied sufficiently often to keep the mask moist :—

℞ Creosoti ʒi.
Spiriti ætheris ʒi.
Acidi carbolici ʒii.
Spiriti chloroformi ʒii.
Tincturæ iodi ʒii.

Creosote may also be given by mouth provided the dose is small enough not to interfere with the appetite ; a quarter of a minim, with one grain of potassium iodide, may be given in a teaspoonful of malt three times a day.

Surgical treatment of bronchiectasis, other than lobectomy, is seldom undertaken in childhood, for in the milder cases there is a fair chance of recovery with medical treatment, while the severe cases are too great a surgical risk. Lobectomy should be considered when the disease is confined to one lobe, for although the operation is a severe one, the risk is actually less in children than in adults, and the after-results are very satisfactory. Fawcett[1] has recorded a successful lobectomy in an infant of nineteen months.

PULMONARY ABSCESS

Multiple pyæmic abscesses may be found in the lungs after death from septic conditions such as osteomyelitis. Both lobar and broncho-pneumonia occasionally go on to abscess formation, but the most likely cause is the presence of a foreign body in a bronchus. The aspiration of septic material during operations on the nose and throat is also a cause, and it has been estimated that this complication occurs roughly in one in every 3,000 tonsillectomies. An abscess may occasionally follow when the chest has been needled for a supposed empyema.

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[1] Fawcett, A. W., *Brit. Med. Journ.*, 1941, June 7th.

The symptoms of pulmonary abscess resemble those of empyema, and consist of an irregular fever, cough, sweating, and loss of flesh. When the abscess is superficial the physical signs are also likely to suggest an empyema, and this will seem to be confirmed if pus is withdrawn through an exploring needle. The presence of a fringe of fine crepitations surrounding the supposed empyema should bring to mind the possibility of a pulmonary abscess, although the same findings may be met with in an interlobar collection of pus. Rupture of the abscess into a bronchus may occur, and then the sudden expectoration of a large amount of pus will not only indicate the correct diagnosis but may also lead to cure.

When abscess of the lung has been diagnosed, spontaneous rupture should not be waited for, but the abscess should be opened and drained. When the abscess has already ruptured, treatment must be determined by the circumstances of the case. A small solitary abscess which has ruptured spontaneously can sometimes be treated by keeping the child in a position that will afford a proper drainage. The discharge of pus then ceases in two or three days, and the child makes a rapid recovery. Alternatively the abscess cavity may be drained through a bronchoscope, or if situated superficially it may be wiser to drain the cavity through the pleura.

Gangrene of the lungs arises from causes identical with those of pulmonary abscess, together with a super-imposed infection by anaerobic organisms. The characteristic symptoms consist of a gangrenous acrid smell to the breath, and the expectoration of dark brown, bloodstained, putrid material, containing shreds of pulmonary tissue. The constitutional effect is one of severe depression, with rapid pulse and hectic fever. Death generally ensues within a few days. When gangrene is due to a foreign body and the foreign substance can be removed with a bronchoscope, recovery is possible.

PULMONARY COLLAPSE (ATELECTASIS)

At the time of birth the establishment of breathing should cause both lungs completely to expand. Occasionally, however, either the whole or a portion of a lung fails to expand, and to this the term congenital atelectasis is given. A more complete account of this is given on p. 10.

An acquired collapse of the lung is common in infancy, and may arise from various causes. The bronchi are so small that a relatively small amount of secretion is capable of blocking the lumen, and accordingly a considerable amount of collapse may arise during both bronchitis and broncho-pneumonia. At autopsy, collapse is most obvious at the bases of the lungs and along their posterior borders. Any debilitating illness, such, for instance, as diarrhœa or marasmus, may so enfeeble an infant that respirations become shallow and fail to expand the lung properly, while rickets, by softening the thoracic cage, may also be a contributory cause. Pulmonary collapse, as shown by indrawing of the intercostal spaces and xiphisternal notch, is often a feature of congenital heart disease in infancy, so much so that the appearance of the chest is often enough to prompt a careful examination for a congenital bruit. Mention has already been made of the collapsed condition of the left lower lobe, which gives radiologically a triangular shadow behind the heart, and which is likely to progress to atelectatic bronchiectasis.

Physical signs of acquired collapse are often difficult to detect, partly because the collapsed areas are generally small and scattered, and partly because breath sounds from the adjacent lung may be easily heard over the area of collapse. In any instance of unilateral collapse the possibility of a foreign body must be constantly borne in mind.

Appropriate treatment must be directed towards the cause of the collapse, whether it be a foreign body, bronchitis, etc. Cases that are due to states of weakness and exhaustion require stimulant treatment, and such measures as a warm mustard bath or inhalations of oxygen and CO_2 should be used. A steam tent is absolutely contra-indicated, for the inhalation of a moist atmosphere is only likely to clog still further the air passages. Atropine is sometimes useful in checking bronchial secretion, and should be given in doses sufficient to cause dryness of the mouth.

Massive Collapse of the Lungs

In this condition the whole of a lung, or one complete lobe, becomes rapidly deflated without any obvious bronchial obstruction. In childhood the condition is rare, but may be met with in post-diphtheritic paralysis affecting the diaphragm, or may sometimes occur after abdominal operations. In such cases the collapse appears as a rule within a day or two of operation, the temperature rises two or three degrees, and over the affected area of lung the percussion note becomes dull, air entry is diminished, and breath sounds are tubular. The signs are similar to those of lobar pneumonia with the notable exception that the heart and mediastinum are displaced towards the collapsed lung. The prognosis is as a rule good. After a week to ten days the lung gradually re-expands. The only treatment required is rest.

Actinomycosis of the Lungs

Actinomycosis of the lungs is a rare affection. The symptoms are likely to suggest an empyema or possibly a malignant growth of the lung, the recognition of the true nature of the condition depending on the recovery of the fungus either from pus removed through an exploring needle or from sputum. Recovery has occasionally followed treatment with large doses of potassium iodide, the dose being worked up to as much as a drachm or more three times a day. Lipiodol injections into the lung have also been used successfully. In cases which recover the course is a long one, but more usually the child rapidly wastes and dies from exhaustion.

Intra-Thoracic New Growths

Intra-thoracic new growths are rare in children. Simple growths such as fibromata or dermoid cysts may arise in the mediastinum, and give evidence of their presence by pressing on surrounding structures. Dyspnœa, cyanosis, a brassy cough, œdema of the face and arms, and dysphagia, may all be produced in this way. Malignant tumours in the thorax are usually sarcomatous and arise as secondary deposits. They show a predilection for growing from the pleura, where they appear as multiple sessile or stalked purplish growths, and are likely to give rise to pleural effusion. Primary malignant growths are very rare, and take the form of lymphosarcoma growing from the mediastinal glands.

Congenital Cystic Disease of the Lung

Congenital cystic disease of the lung is fortunately a rare condition. The cysts may be single or multiple, and should properly be regarded as arising from the bronchi, as they are lined with bronchial epithelium.

The single or " balloon " cyst is met with in early infancy. The cyst communicates with the bronchus by a valvular opening which allows inflation to take place, but the air cannot escape, with the result that the cyst rapidly distends until it occupies the whole of one side of the chest, compressing the lung on that side and displacing the heart and mediastinum far over to the opposite side. The infant suffers from severe dyspnœa and cyanosis, and examination gives physical signs which correspond to a pneumothorax under tension. An X-ray (see Plate VII) at first suggests a pneumothorax, but thin strands of tissue may be seen crossing the air-space. Withdrawal of air may give temporary relief, but the cyst soon refills, or if the thin cyst wall is torn, as may easily happen, a pneumothorax will result. Death occurs within a few weeks of birth (see Plate VII).

Multiple cysts may be few in number, or the whole of a lobe or a lung may be riddled with small cysts (honeycomb lung). There may be no symptoms until infection occurs, and this may be postponed for some years, but thereafter the symptoms and signs are those of bronchiectasis. Treatment consists of excision of the affected area if its distribution makes this possible.

DISEASES OF THE PLEURA

The two commonest forms of pleurisy in childhood are: (1) Empyema, and (2) pleurisy with serous effusion. The latter is almost always tuberculous and is dealt with in the section on intra-thoracic tuberculosis. A passive transudate of fluid into the pleura may accompany nephritis or cardiac failure just as in adults. Dry pleurisy, indicated by fever, pain, shallow respirations, and audible friction sounds, often occurs with lobar pneumonia and broncho-pneumonia. That it may also occur in association with pulmonary tuberculosis and rheumatic carditis is shown not infrequently at autopsy by the presence of pleural adhesions, although there may have been but little evidence of it during life.

An epidemic form of pleurisy has been described which chiefly concerns children.[1] The onset is brisk, with fever, shortness of breath, pain often localised near the xiphisternum, and after a few hours the presence of pleural frictions confined perhaps to a small area. The condition subsides within a week without complications. The illness has also been called "Bornholm" disease, after the Island of Bornholm in the Baltic, where it is frequently met with.

EMPYEMA

Empyema in childhood is most likely to be met with in the early years. In a series of 100 consecutive cases at The Hospital for Sick Children, 49 were under three years of age. Boys are affected a little more often than girls. Lobar pneumonia accounts for roughly three-quarters of the cases, and therefore, as one would expect, the pneumococcus is the causal organism in at least 75 per cent. The next most common organism is the streptococcus, streptococcal cases being met with when the empyema follows broncho-pneumonia. Occasionally a mixture of two or more organisms is obtained from the pus.

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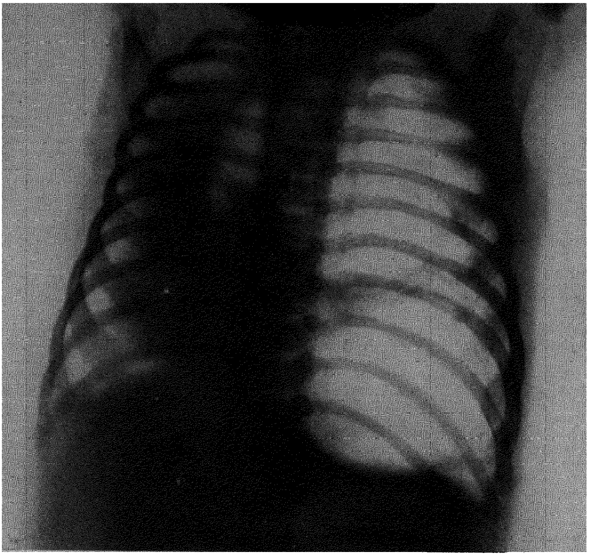
[1] W. N. Pickles, *Brit. Med. Jour.*, 1933, *ii.*, 817.

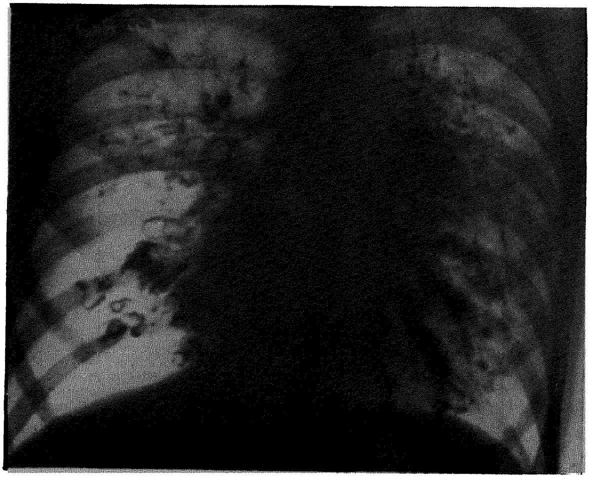
Symptoms. The symptoms vary in their severity according to the amount of the effusion. As a rule there is a history of pneumonia followed by a recrudescence of fever two or three days after the crisis. Less often the presence of an empyema may be suspected before the pneumonia has reached its crisis. In other cases there is no history of a preceding illness, the child is brought for examination on account of cough, loss of weight, and increasing pallor of a few weeks' duration, and signs of an empyema are unexpectedly discovered. Breathlessness is a common symptom, and is likely to be made worse when the child lies on the sound side of the chest, so much so that one may be told that since the onset of his illness the child prefers to lie only on one side, or he may only be able to breathe comfortably when sitting up. Cough is usual, and is frequent, shallow, and unaccompanied by sputum. Pain is a very variable symptom, it may be present in extensive effusions owing to pressure on adjacent structures, but a feeling of discomfort amounting almost to suffocation is more common than actual pain. The temperature is also very variable, and is not to be taken as a guide to the presence or absence of pus. As a rule it lies between 99° and 101°, but may run up to 104° or higher.

The appearance of a child with empyema is often suggestive. The complexion has a sallow earthy tint with underlying pallor, and considerable sweating often occurs in the early stages, although if an empyema is allowed to go unrecognised for three or four weeks the skin becomes dry, and the child wastes rapidly. Clubbing of the fingers appears after two or three days, appearing at first as a heaping-up and shininess of the fingers just behind the nails, and becoming gradually more marked until the empyema is relieved. Examination of the chest shows some impairment of movement on the affected side, and if the exudate is very extensive there may be bulging of the intercostal spaces and even œdema of the chest wall, but this is not common. The apex beat is as a rule displaced away from the side of the empyema, the degree of displacement depending partly on the amount of pus

PLATE VII.



Congenital " balloon " cyst of the left lung in an infant aged nine weeks. Note the severe mediastinal displacement, the low position of the diaphragm, and the strands of tissue crossing the cystic space.



X-ray of a boy aged ten years, showing multiple cystic disease of the lungs, more marked on the right side.

PLATE VIII.

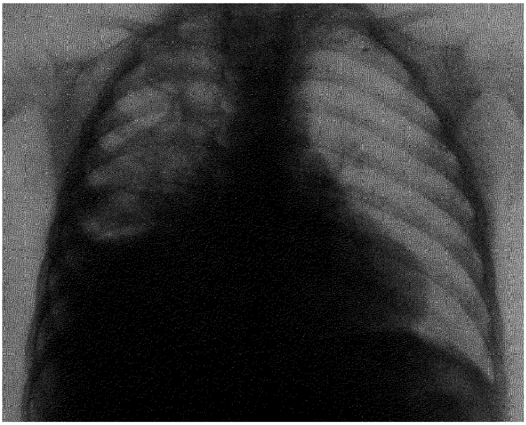


FIG. A. Right-sided empyema in a baby aged eleven months. The heart is displaced to the left.

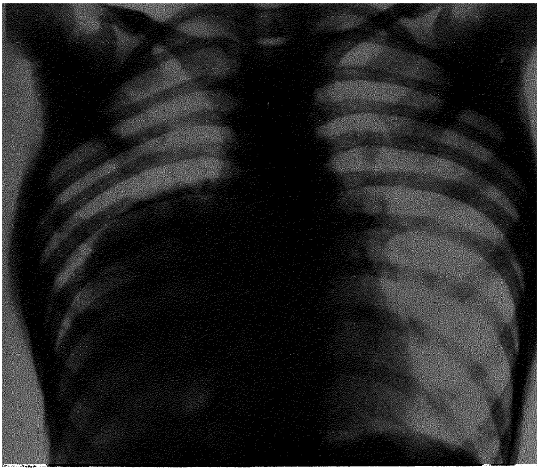


FIG. B. X-ray of a girl aged ten years, showing a large interlobar collection of pus on the right side. Note the absence of any displacement of the heart or trachea. The case is described on p. 374.

and partly on its position. Thus there may be scarcely any displacement with a small empyema, nor when the pus is locked away in the inter-lobar fissure or at the apex of the lung. The trachea is displaced with the heart. The percussion note over the empyema is stonily dull, in fact so dull as to be characteristic of the presence of fluid, and dull enough to be distinct from the note obtained over an area of consolidation of the lungs. Not infrequently the percussion note above the level of fluid has a tympanic quality. Auscultation typically shows an absence of breath sounds and also an absence of adventitious sounds, indeed the presence of a few crepitations is more suggestive of unresolved pneumonia than an empyema. An empyema may however be present even when the breath sounds are tubular, in fact it must be stressed that in a child tubular breathing does not exclude an empyema. Tubular breathing is also often heard above the level of fluid, giving an area of bronchial breathing over the middle or upper third of the chest, and this should always prompt an examination at the base of the chest for signs of empyema. Vocal resonance is seldom of value in a child because of the weakness of the voice, but if the child is crying the voice sounds will be less well conducted over the area of fluid. X-ray examination shows an opaque shadow in the position of the empyema, and is particularly helpful when the empyema is loculated to some small part of the pleural surface.

There is generally a heavy leucocytosis, the white cells numbering between 25,000 and 50,000 per c.mm. with a relative preponderance of polymorphs up to about 80 per cent. The white cell count is generally higher in pneumococcal than in streptococcal cases.

Varieties. Bilateral empyemata occasionally occur after double pneumonia. When pus has been obtained from one side of the chest it is easy to overlook the collection on the other side, but if the temperature fails to settle and the child makes no progress after one side has been adequately drained, the possibility of a bilateral empyema should be borne in mind. Such cases present great difficulty, because as soon as one side of the chest is open it becomes difficult to interpret physical signs. In any doubtful case the opposite side of the chest should be examined with an exploring needle.

As a rule the pus collects freely at the base of the pleura, but may sometimes become walled off in different situations. Particular difficulty is experienced when the pus is loculated at the apex of the lung, or between the pericardium and the lung, or between the base of the lung and the diaphragm. A collection of pus in the latter situation may give rise to a good deal of abdominal pain, or the pain may be referred to the shoulder, and if the collection of pus is extensive the liver or spleen may be displaced downwards. Probably the most frequent variant of ordinary empyema is an interlobar collection of pus, and in such cases the physical signs are often so modified as to be very misleading. There may be a strip of deflated lung between the chest wall and the empyema, which makes the dullness on percussion less absolute, while from the compressed lung a few crackling crepitations may be audible. It is to be remembered also that pus loculated in this way may produce hardly any mediastinal displacement. In these cases an X-ray examination is often of great help.

The lower figure on Plate VIII shows a large interlobar empyema which only just reached to the chest wall, and gave dullness and absent breath sounds over an area that could be covered with a half-crown. It will be noticed that there is air-containing lung between the chest wall and the empyema, and also between the lower border of the empyema and the diaphragm.

Diagnosis. Although as a rule the diagnosis is simple enough, it may be most baffling, especially when the pneumonia has passed unrecognised, or when the empyema forms before the crisis of the pneumonia (synpneumonic empyema), or when the empyema is loculated. Conditions which may simulate an empyema are pulmonary abscess, massive collapse of a lung, and rarely a lobar pneumonia which is so massive that the bronchioles are filled with secretion and breath sounds are therefore absent. It is often difficult to decide whether a pleural effusion is serous or purulent until the exploratory puncture has been done.

The ultimate diagnosis of an empyema rests on the discovery of pus with an exploratory needle, and whenever reasonable doubt exists the pleura should be explored in this way. The proper place to explore the chest is wherever the percussion note reaches its maximum dullness ; in the usual type of case this will be in the eighth or ninth space just below the inferior angle of the scapula. As a rule local anæsthesia is all that is required, but if there is likely to be difficulty in finding the pus, as, for example, when seeking an interlobar collection, it is better to have the child under a general anæsthetic. The accidents which may follow an exploratory puncture include : puncture of the lung with resulting pneumothorax, the formation of an abscess in the lung, surgical emphysema, and cellulitis of the chest wall.

Prognosis. The mortality from empyema is greatest in young children. Of 100 consecutive cases at Great Ormond Street, the mortality in 27 children under two years of age was 66 per cent., while in 44 children between two and five years of age the mortality was 14 per cent., and in 29 others between five and twelve years only 6 per cent. were fatal. Age is a more important factor in determining the outlook than is the particular infecting organism, but pneumococcal cases are more favourable than streptococcal. Staphylococcal empyema is rare, but carries a high mortality. When an empyema develops before the crisis of a pneumonia the outlook is made worse.

As a rule, when an empyema has been drained it takes a month or six weeks before the child can get up, and it may be several more months before the lung is properly expanded. When an empyema remains untreated, various sequelæ may result. The organism may gain the blood stream and produce septicæmia, or the empyema may rupture into a bronchus and give rise to the expectoration of much pus ; occasionally the empyema undergoes in this way a spontaneous cure, but generally the chest has to be opened and drained on the ordinary lines. Or the pus may penetrate the pleura and burrow externally to point between the ribs or even more distant parts (*empyema necessitatis*). No doubt a small empyema may become inspissated and the pus be absorbed, but severe fibrosis of the underlying lung is then almost certain to follow. Metastatic infections may develop in other parts, *e.g.*, meningitis, cerebral abscess, peritonitis, arthritis or pericarditis. Of 100 fatal cases of suppurative pericarditis in children, Poynton [1] found that 60 were associated with empyema.

Treatment. When an empyema forms before the crisis of a pneumonia the pus should be aspirated daily or every other day until three or four days after the crisis, when more thorough drainage can be instituted. Aspiration is also the method of choice when dealing with a streptococcal empyema, repeating it until such time as the pus has changed from a thin and perhaps blood-stained fluid to one of thicker consistency, and the same may be said of a pneumococcal empyema when the pus is at first thin, although as a rule in this type the pus is thick at the time of the first exploration.

It is to be remembered that there is every advantage in waiting till the pus is thick before proceeding to drain the empyema, but as soon as this has come about the correct treatment is to ensure adequate drainage. The usual method is to resect a portion of a rib and to insert a rubber tube, drainage continuing for about a week or ten days before the tube is removed. The main advantage of this method is that thorough drainage is ensured, and in children over two or three years of age it answers on the whole as well as any other. Osteomyelitis of the rib is an occasional complication, and great care must also be taken to prevent secondary infection of the wound, which, when it occurs, always delays the healing of the sinus.

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[1] Poynton, F. J., *Brit. Med. Jour.*, 1908, *ii.*, 365.

The high mortality in young children has led to the use of other methods. Simple incision and drainage through an intercostal space without rib resection has the advantage of speed and the avoidance of any shock caused by cutting through the bone, but has a disadvantage inasmuch as the ribs of a small child are so close together that adequate drainage is difficult. Various methods of continuous suction drainage have been attempted, the underlying principle being to have an air-tight drainage at the chest wall with a tube leading to a bottle in which a negative pressure is maintained by attachment to a syphon. This method has the great advantage of only requiring a local anæsthetic, the drainage can be done with the minimum of disturbance and without the child being moved from its bed, daily dressings are avoided, and the pus can be drained off as slowly as one desires ; but, on the other hand, it is difficult to tell when the cavity has been emptied, and there is often a technical difficulty in keeping the opening at the chest wall air-tight. Continuous suction drainage is particularly valuable in small children, but it is a method that requires expert handling. Another method which has proved useful is the insertion of two self-retaining catheters into the chest, using one for drainage, while the other is employed to irrigate the pleural cavity with an antiseptic solution such as Dakin's solution. Further details can be obtained from the two references given below.[1]

When an empyema follows a pneumonia that has been treated with one of the sulphanilamide preparations, the characters of the fluid may be modified ; the pus may remain thin, a proportion of the cells may be lymphocytes, and culture may be sterile. Even so, once pus has formed, surgical drainage should be carried out, and sulphanilamide treatment should be discontinued.

When the pus has been evacuated every effort must be made to get the collapsed lung to re-expand fully. Fresh air and a nourishing diet are important, and systematic exercises are of value. A change of air to the seaside will often bring about the most remarkable improvement in a child whose empyema sinus has failed to heal after weeks in a ward. Exposure of the sinus to ultra-violet radiation is sometimes beneficial. If the sinus is very persistent, an autogenous vaccine is worth a trial, but in long-standing cases some form of thoracoplasty will be needed.

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[1] Browne, Denis. *Lancet,* 1930, *ii.,* 733.
Thomson, M. L. *Lancet,* 1934, *ii.,* 1435.

Pneumothorax

Pneumothorax is a rare event in childhood. Most of the cases arise in association with tuberculosis of the lungs, and are due to the breaking down of a caseous area through the pleura. Frequently fluid is present in the pleura as well as air—a tuberculous pyopneumothorax. More rarely pneumothorax occurs from the rupture of a vesicle during severe coughing bouts in whooping-cough, and it has been recorded as a complication of broncho-pneumonia. Traumatic pneumothorax after accidents or as a result of exploratory puncture of the chest is also rare, and with rest and immobilisation of the affected side the traumatic cases usually recover after a few days. Spontaneous pneumothorax has occasionally been recorded in newborn infants, and has been attributed to respiratory obstruction, infection, and congenital pulmonary defects. The main symptoms are increasing dyspnœa

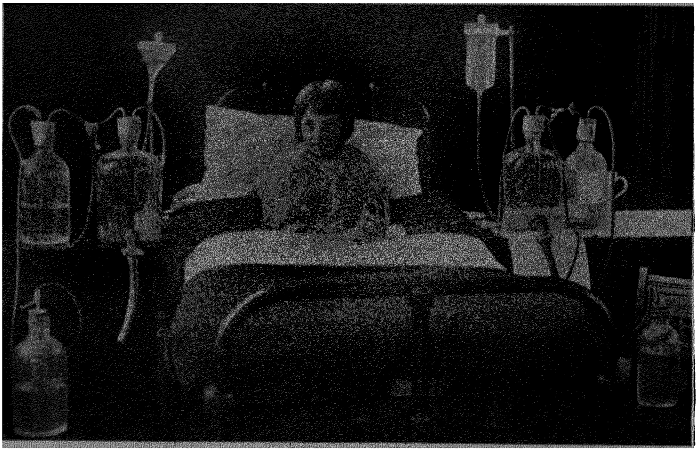

FIG. 56. Bilateral suction drainage in a girl aged six years with double empyema. (By courtesy of Dr. M. L. Thomson and the *Lancet.*)

and cyanosis. The condition must be distinguished from congenital cystic disease of the lung (p. 370).

The symptoms and signs of pneumothorax in a child are the same as in an adult. In those cases which complicate underlying disease of the lungs the occurrence of pneumothorax renders the prognosis very grave. Treatment consists of complete rest combined with small doses of opium, but if the tension in the pneumothorax becomes so great as to produce serious symptoms from the displacement of the heart, such as cyanosis, breathlessness, or restlessness, the chest should be explored and the air gradually released under control.

CHAPTER XVI

INTRA-THORACIC TUBERCULOSIS

It is very difficult to arrive at figures which indicate with any accuracy how common thoracic tuberculosis is among young children, but that it is a most frequent cause of death during childhood cannot be doubted. In the writer's series of 400 consecutive autopsies made at Great Ormond Street, tuberculosis in all its forms accounted for 17 per cent., and of the deaths from tuberculosis the primary site of infection was in the thorax in 67 per cent.—a figure which compares closely with the series of tuberculous children examined at post-mortem by Still, who found that intra-thoracic infection accounted for 63 per cent., and with Blacklock's figure of 61 per cent.

The infection almost invariably reaches the lungs by inhalation of the bacillus from droplet infection, spread as a result of persons already infected coming into the immediate vicinity of the child. So great is the probability of a child becoming infected if he comes in contact with phthisical adults that with such a history it is safe to assume that the child has become infected. There is no evidence that a child can inherit a peculiar liability to tuberculosis ; the frequency of tuberculosis among infants who are born into tuberculous households is due to the great risk they run of direct infection.

Intra-thoracic tuberculosis in young children differs from the disease as seen in adults in two important ways. Firstly there is pre-eminently a tendency for the lymphatic glands in the mediastinum to become involved at an early stage, and indeed to an extent often quite out of proportion to the amount of disease in the lungs, and secondly a far greater tendency in childhood for the infection to become disseminated in the blood stream, leading to a fatal issue either from meningitis or miliary tuberculosis.

During childhood the age incidence of intra-thoracic tuberculosis as judged from post-mortem records rises rapidly towards the end of the first year, and reaches its peak during the second and third years. Thereafter the number of fatal cases steadily declines. A similar age incidence also characterises other severe forms of tuberculosis, such as abdominal, meningeal, or miliary infection. There are two diseases which stand out as predisposing to the development of thoracic tuberculosis, namely measles and whooping-cough. They are both liable to be complicated by a broncho-pneumonia which resolves slowly and leaves the mediastinal glands swollen and congested for several months, a state of affairs which not only makes the lungs a most ready nidus for tuberculosis, but also makes the re-activation of an old focus very likely.

Pathology. Infection by the tubercle bacillus gives rise in the body to two distinct processes ; one is the gradual development of immunity by which recovery ultimately comes about, the other is the development of hypersensitivity or allergy to the protein liberated from the dead organisms (tuberculin). Whether the second process assists the body in overcoming the infection is open to question, but at all events the experiments of Rich[1] have shown that the two processes bear no relation to each other, the presence or absence of the one giving no indication as to the state of the other. When infection takes place in a young child there is no preformed immunity to counter it, which may help to explain the ease with which general dissemination occurs at that age. On the other hand, the allergic response plays a more important part in the clinical picture in children than in adults.

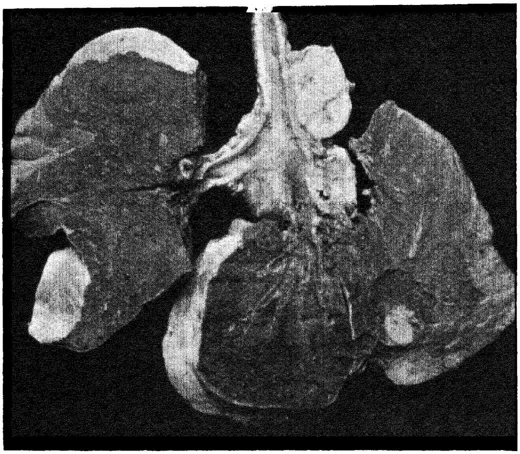

FIG. 57. The lungs of a child showing a primary focus of tuberculosis at the base of the left lung, with secondary caseation of the mediastinal glands. (From the museum of The Hospital for Sick Children.)

The inhalation of the tubercle bacillus into the lungs gives rise to a definite sequence of pathological events. The organism is first inhaled into a small bronchiole and there penetrates the mucosa, where the reaction to its presence leads to the formation of a tubercle. This undergoes the ordinary sequence of changes, namely caseation, and eventual healing by calcification, or liquefaction and discharge of its contents into a bronchiole to form a small cavity. A local spread to neighbouring parts of the lung is quite unusual in children. The importance of this " primary focus " in the lung as the initial lesion of intrathoracic tuberculosis in childhood was noted by Parrot in 1876 and has been emphasised by Ghon,[1] who in a series of 184 postmortems on children with thoracic tuberculosis was able to demonstrate the primary focus in 92 per cent. The primary focus may vary in size from a pin's head to a hazel nut, and is usually much too small to be noticed during clinical examination of the chest, although if calcified it may show on the X-ray film. Such a calcified focus has been found as early as ten months of age. As a rule the primary focus is situated in the vicinity of the pleura, and is more often in the right lung than the left, and usually in the upper lobes. Although there is usually but one primary focus, there may be several.

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[1] Rich, A. R., *Lancet*, 1933, *ii*, 521.

The next stage of the disease consists of a spread of infection from the primary focus along the lymphatics to the mediastinal glands. It used to be thought that the mediastinal glands could be infected by tubercle bacilli travelling directly from the abdominal lymphatics, or from the cervical glands, or by a passage through the lungs without leaving any evidence of a pulmonary lesion, but in the light of Ghon's work these other routes of infection must be regarded as very uncommon. It is probable that about six to ten weeks elapses between the initial infection and the development of tuberculosis in the mediastinal glands. It is also towards the end of this interval that the child develops a state of hypersensitivity to tuberculin, which can be demonstrated by obtaining positive skin reactions to the Vollmer patch test or following an intradermal injection of tuberculin, and may manifest itself clinically by the appearance of phlyctenular conjunctivitis, or erythema nodosum. It may be pointed out that although these hypersensitive phenomena are all benign conditions, they are of great practical importance inasmuch as they stand as evidence of early tuberculous infection. Erythema nodosum is more fully considered on p. 636. In an enquiry into

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[1] " Primary Lung Focus and Tuberculosis in Children." By Anton Ghon, trans. by Dr. Barty King, London, 1916.

1,392 children with phlyctenular conjunctivitis, Sorsby [1] found positive tuberculin skin reactions in 84 per cent., and there was radiological evidence of intrathoracic tuberculosis in 72 per cent. The importance which attaches to these two investigations in the diagnosis of intrathoracic tuberculosis in children, and the customary paucity of physical signs, is also brought out by this enquiry, for clinical evidence of tuberculosis was present in only 6·4 per cent.

The mediastinal glands first to be affected are those which lie along the main bronchus of the lobe containing the primary focus. Only one or two glands may be affected, and these may go through a stage of caseation and healing by calcification, or the infection may spread from gland to gland to infect the big gland at the bifurcation of the trachea, and so cross to those on the opposite side or spread upwards to involve the glands alongside the trachea, until eventually a large conglomerate mass of caseous glands surrounds the trachea and main bronchi. Occasionally, infection may pass from the tracheo-bronchial glands up to the lower cervical glands, and rarely to the upper abdominal glands, but, if it spreads at all, it is much more likely to travel directly into the blood stream *viâ* the thoracic duct or the right lymphatic duct.

The majority of children with tuberculous mediastinal glands undoubtedly recover, but the disease may progress in various ways. A gland may rupture into a bronchus, and its contents be inhaled over part or the whole of a lobe or into both lungs ; what happens then will depend upon the dose of organisms inhaled, their virulence or viability, and the presence or absence of allergy. If the dose is heavy and the organisms are virulent a tuberculous broncho-pneumonia will result and will be speedily fatal. If on the other hand the organisms are for the most part dead but the child is already sensitive to tuberculin, a benign tuberculous pneumonia will result, which will run a protracted course to eventual recovery (epituberculosis). We must also expect to see examples intermediate between these two types. Or the glands may leak into the blood stream, with effects which will again depend on the factors just mentioned. Thus if the number of organisms is small, isolated foci of tuberculosis will develop in other parts of the body, such as in the spleen, brain, joints, or kidney, while if the blood stream is flooded with organisms, tuberculous meningitis or generalised miliary tuberculosis will result. Occasionally a chronic form of miliary tuberculosis occurs, from which the child may recover, or, after repeated exacerbations, may succumb. Here we are probably dealing with an intermittent discharge into the blood stream of relatively small numbers of organisms. In a little girl observed by the writer the rupture of a caseous gland into the descending aorta caused the immediate death of the child. Rupture into the œsophagus has also been recorded.

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[1] Sorsby, A., *Brit. Jour. Ophthal.*, 1942, 26, 159 and 189.

Symptoms. It has already been said that the primary lung focus of tuberculosis is generally much too small to be detected on clinical examination, but the other stages of the disease have a clinical picture which must now be described under the following headings :—

Tuberculosis of the Mediastinal Glands.
Tuberculous Broncho-Pneumonia.
Miliary Tuberculosis.
Epituberculosis.
Tuberculous Pleurisy.

Tuberculosis of the Mediastinal Glands

There is nothing absolutely diagnostic about any of the symptoms of enlarged mediastinal glands, and the diagnosis is almost always a difficult matter, being generally based on the summation of several suggestive signs combined with the help of X-ray examination.

One of the most important symptoms is cough. This is as a rule harsh and dry, and may sometimes have a paroxysmal character. There is no visible sputum, although tubercle bacilli can sometimes be recovered from a gastric lavage, indicating that a certain amount of infected sputum is being produced and swallowed. There is also likely to be a history of a failure to gain weight, loss of appetite, listlessness, and there may be a low irregular fever ranging between 99° and 100° F. An increase of downy hair on the back and arms has been described as indicating tuberculous infection, but it is not an uncommon finding in children who suffer from any prolonged infection such as unresolved pneumonia or a chronic empyema.

During the first eighteen months of life, when the chest is so small that there is little room for the mediastinal glands to enlarge, the trachea or bronchi may become compressed, and then the cough may sound metallic or brassy, and inspiration may be accompanied by stridor. A stridor which comes on gradually some months after birth, but which persists, should always arouse suspicion of tuberculous mediastinal glands. In some cases the compression leads to collapse of a portion of a lung ; in others, air can be drawn past the obstruction but cannot escape, leading to obstructive emphysema (see Fig. 58).

The signs on examination of the chest are often obscure, and no particular one can be relied upon as diagnostic of enlarged glands. Inspection may show dilated veins coursing along the first and second intercostal spaces, but these are only of significance if they are of recent appearance. More often they have been present from birth. On percussion there may be some impairment of note close to or under the manubrium, and there may be also some loss of resonance between the scapulæ and the spine, but as a rule these percussion signs are more suggestive of a tuberculous consolidation of the roots of the lungs than of

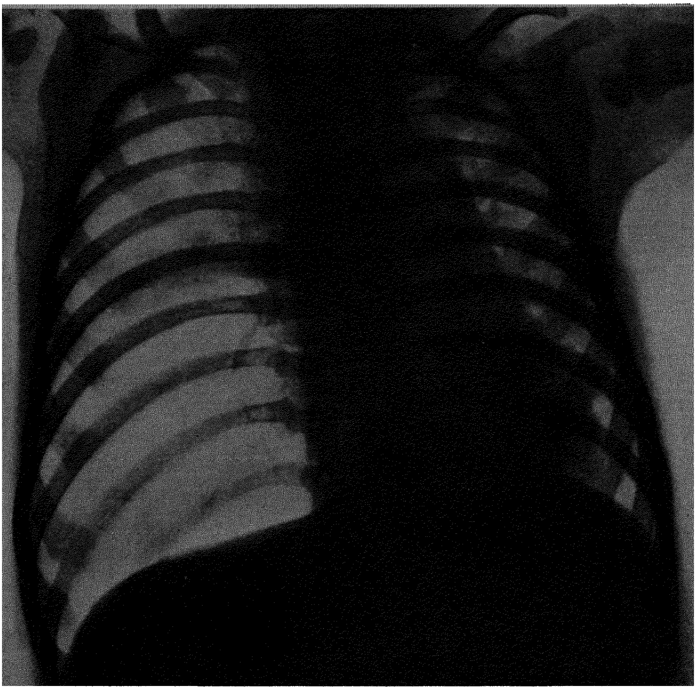

FIG. 58. X-ray of a girl aged ten months, showing obstructive emphysema of the right lung, especially in the lower half. This was the result of bronchial compression by tuberculous mediastinal glands.

enlarged mediastinal glands. Eustace Smith's sign consists of a systolic murmur heard under the inner end of the right clavicle when the head is thrown back, but it is so frequently and easily obtained in thin or debilitated children that little reliance can be placed on it. More important is the occasional evidence of patches of diminished air entry, which are most likely to be found high up in one or both axillæ, or stretching down posteriorly from the hilum to the base of the lung close to the vertebral column. Occasionally crepitations may be heard over the root of one or other lung, and are due either to tuberculous bronchitis (hilum catarrh), or to a breaking down of a focus in the adjacent lung with cavity formation. When the latter occurs, a moist cough, irregular fever, and loss of weight will also be present. Probably the most valuable sign is that described by d'Espine, which is obtained by listening to the breath sounds over the thoracic spines. Normally a tracheal quality to the breath sounds, and whispering pectoriloquy, can be heard as far down as the second thoracic spine, that is to say down to the level of the bifurcation of the trachea, but when the gland in the bifurcation is enlarged the tracheal quality may be conducted as far as the fourth or fifth thoracic spine.

Clinical examination is much aided in these difficult cases by good X-ray photographs of the chest. In the usual antero-posterior view the mediastinal glands shelter behind the base of the heart and large vessels, and even quite large glands may be obscured. This can to some extent be overcome by X-raying the chest in the lateral position, when enlarged glands may show as opacities in the posterior mediastinum at about the level of the bifurcation of the trachea (see Plate IX).

Mediastinal glands may be enlarged for various reasons. They are commonly enlarged in measles, whooping-cough, and active rheumatic heart disease, and always in broncho-pneumonia, while lymphadenoma and lymphosarcoma are rarer causes. It follows that having diagnosed enlarged glands in the mediastinum it still remains to show whether they are tuberculous. Pin-points of calcification in the X-ray film would be conclusive, a history of contact with a tuberculous relative would also be strong presumptive evidence, while a history of cough arising insidiously and persisting over several months must always be suggestive. Tuberculin skin tests[1] are a useful adjunct to diagnosis in young children.

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[1] The technique of these tests is described on p. 248.

Tuberculous Broncho-pneumonia

Occasionally a caseous focus in a mediastinal gland or in the lung discharges its contents into a bronchus, and the bacilli become freely inhaled over the neighbouring area of lung or even over both lungs to give rise to tuberculous broncho-pneumonia, or the same result may come about from a heavy re-infection by inhalation from some exogenous source. The clinical distinction between tuberculous and non-tuberculous broncho-pneumonia is at times very difficult, especially in young children. A frequent cough, listlessness, flushing of the cheeks, sweating, rapid loss of weight, and hectic temperature are generally very evident. The signs are those of an ordinary broncho-pneumonia, and consist of an impaired percussion note, fine metallic crepitations, and sometimes patches of tubular breathing. X-ray examination shows a fluffy opacity in the lungs very similar to that seen in non-tuberculous broncho-pneumonia. The outlook in these cases is as a rule bad, and after a course of a few weeks or months the children die either from exhaustion and toxæmia, or from a general or meningeal dissemination of infection. Exceptions occur, however, in which the illness, though prolonged, is mild, and recovery eventually comes about.

Tuberculous pneumonia rarely assumes a massive form, in which the whole of a lung becomes converted into a caseous mass. The onset and course is at first closely similar to a lobar pneumonia, but instead of a termination by crisis the illness continues for a few weeks, the temperature becomes hectic, and the child eventually succumbs to exhaustion. Cases of pneumonia migrans (p. 353) are very likely to be confused with this form of tuberculosis, but the orderly spread of the pneumonia from lobe to lobe serves as a distinguishing feature.

Chronic pulmonary tuberculosis of the type met with in adults, and either fibro-caseous or ulcero-cavernous, is seldom seen in childhood, and is very rare before seven or eight years of age. Such an area of disease probably arises as a result of direct extension round the primary focus of Ghon, although some have considered that the lung may become re-infected by a retrograde lymphatic spread from the mediastinal glands, the lung tissue becoming involved at some relatively distant spot. It may also be caused by a fresh infection from some outside source. In this form of tuberculosis the upper lobes are usually involved, although not so frequently in children as in adults. Hæmoptysis is also much less common. Gradual recovery with fibrosis is possible even when cavities have formed, but usually the disease extends

PLATE IX.

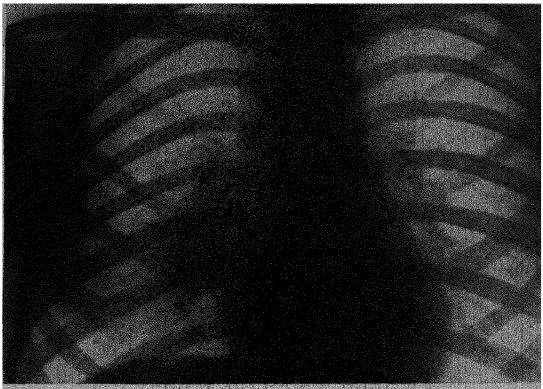

Fig. A. Tuberculous glands at the root of the right lung. Bo aged ten years.

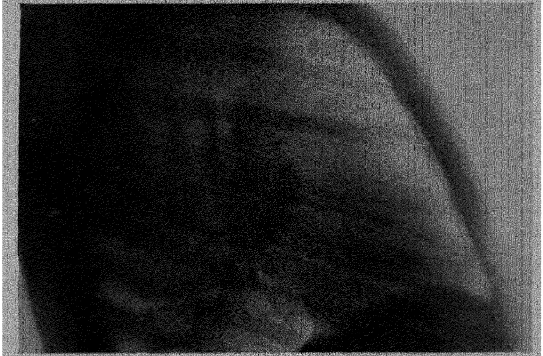

Fig. B. Lateral view to show enlarged glands round the bifurcation of the trachea. Same child as Fig. A.

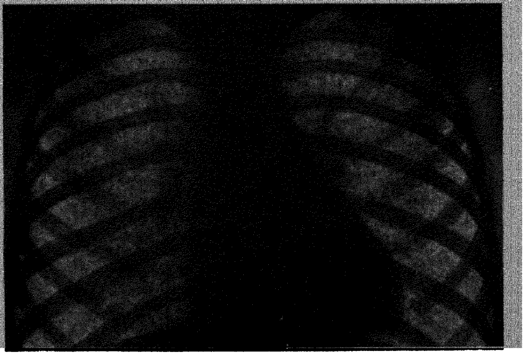

Fig. C. X-ray of a girl aged eleven years showing miliary tuberculosis of the lungs. (By courtesy of Dr. Bertram Shires.)

[*To face p.* 386.

PLATE X.

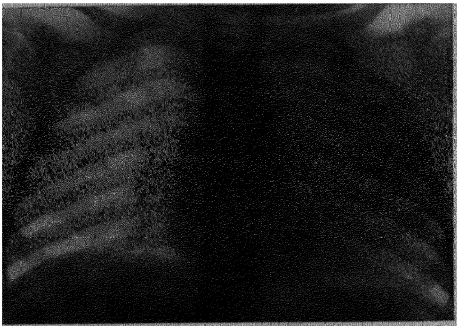

Fig. A. Boy aged four years. X-ray taken on December 31st, 1928, showing epituberculosis of the left upper lobe.

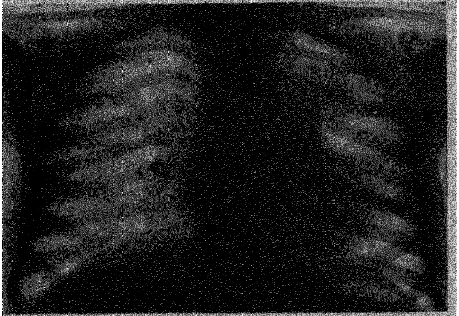

Fig. B. Same case as Fig. A. X-ray taken on April 10th, 1929, showing resolution taking place from the periphery of the left upper lobe.

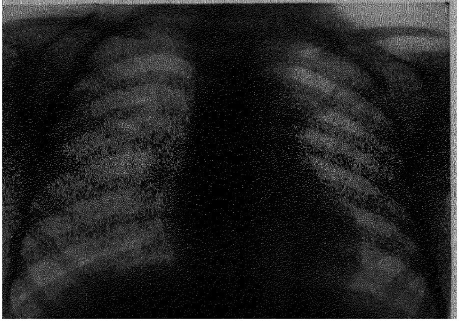

Fig. C. Same case as Fig. A. X-ray taken on October 7th, 1929, showing a further stage in the resolution of the left upper lobe. Six years later the boy was in perfect health.

[*To face p.* 387.

rapidly and the child dies from a general dissemination of the infection.

Miliary Tuberculosis of the Lungs

Miliary tuberculosis of the lungs occurs as part of a generalised miliary tuberculosis, and is due to a blood-stream infection from a caseous focus which is usually in the chest or less often in the abdomen. Occasionally at autopsy the condition is almost confined to the lungs, and then may possibly come about by inhalation of a heavy dose of bacilli leaking from a caseous gland directly into a bronchus.

At post-mortem examination miliary tubercles appear on the pleural surface of the lung and on the cut surface as a host of little grey granulations, each about as big as a pin's head. They are also numerous in the spleen, which is usually enlarged, and they can be made out easily on the surface of the liver and kidneys, in fact careful search will reveal them in almost every organ of the body. There is as a rule an accompanying meningitis.

The general symptoms consist of wasting, tachycardia, and a high swinging temperature. Symptoms of lung involvement are sometimes remarkably few, while in other cases there may be a frequent short cough, some degree of cyanosis, and a rapid respiration rate out of all proportion to the signs obtained over the lungs. Examination of the chest may show only a few fine crepitations scattered over both lungs ; there is as a rule no impairment of percussion note nor alteration of the breath sounds.

The diagnosis is made difficult by the scarcity of signs. Considerable help may be obtained from X-ray examination, which shows a fine mottling over both lungs—aptly described as a snowstorm appearance (see Plate IX). Examination of the fundus oculi may show choroidal tubercles, which appear as small circular fluffy white patches hardly as big as the disc, and generally situated close to a vessel. Their presence is absolutely diagnostic of generalised miliary tuberculosis, and therefore in a suspected case a careful examination of the fundi should be carried out.

Acute miliary tuberculosis is invariably fatal, and treatment can only be palliative, any distressing symptoms being relieved with sedatives.

Chronic Miliary Tuberculosis.[1] From time to time evidence is forthcoming to show that miliary tubercles are capable of undergoing fibrosis with healing. The illness then runs a long course which may terminate by recovery, or may slowly advance to a fatal end, or after one or more exacerbations may finish as does the acute form. At post-mortem examination the paratracheal glands are caseous, and it is probable that the condition is caused by the repeated escape from these glands into the blood stream of small numbers of organisms.

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[1] A recent summary of the literature with report of further cases has been made by R. H. Fish, *Arch. Dis. Child.*, 1937, **12**, 1.

Epituberculosis

This is the name given to a benign form of tuberculous infiltration of the lung, which occurs in young children shortly after the initial infection of the lung has taken place.

The history is usually somewhat as follows : The child, probably under five years of age, is brought on account of a cough of a few months' duration, and failure to gain weight. Clinical examination shows an impaired or dull note over an upper lobe (most commonly on the right side), with bronchial breathing and bronchophony. There may be a few crepitations, but as a rule adventitious sounds are wanting. There is no displacement of the heart or trachea. The temperature may be normal, or raised a degree or two. X-ray examination shows a uniformly dense shadow spreading from the hilum over the upper lobe, the lower edge of the shadow being quite well-defined. Further evidence of the tuberculous nature of the condition is likely to be obtained from a history of the child having been exposed to infection through living in contact with a phthisical adult, and tuberculin skin tests are positive. Tubercle bacilli have also been obtained from the gastric washings of these children.

Epituberculosis is remarkable in that here we have an apparently tuberculous consolidation of the lung bearing a good prognosis. The condition lasts for several months, even up to a year, and if during this time a series of X-rays are taken (see Plate X), the lung condition can be seen gradually to resolve, resolution taking place from the periphery towards the hilum, and eventually leaving a small kernel of calcification near the root.

Probably of a similar nature is the fan-shaped area of consolidation which is sometimes seen on an X-ray film, situated at the root of the lung in association with tuberculous mediastinal glands, and spoken of as a " hilum flare." As a rule, under the influence of rest and fresh air these areas slowly resolve.

The diagnosis of epituberculosis is made on the presence of consolidation of an upper lobe, a history of gradual onset, the exposure of the child to a known source of infection, positive tuberculin skin tests, and the X-ray picture. The long history and insidious onset rules out lobar pneumonia. The differentiation from an unresolved pneumonia which is proceeding to fibrosis may present difficulty, but the absence of any history of pneumonia, the homogeneous appearance of the opacity on the X-ray film, the probable absence of moist sounds, and the ultimately good prognosis are features which distinguish epituberculosis.

The precise nature of the changes occurring in the lung are uncertain. Eliasberg and Newland,[1] who first described the condition, regarded it as a non-specific infiltration, probably allergic in nature, around a tuberculous focus, but tubercle bacilli have been obtained by puncture of the solid lung (Spence). Oppenheimer [2] has shown that the intra-bronchial injection of killed tubercle bacilli into an animal previously rendered hypersensitive to tuberculin leads to a series of changes analogous to those of epituberculosis in the child ; in the latter the condition is probably to be accounted for by the discharge into a bronchus from a tuberculous gland of caseous material impregnated with tuberculo-protein and dead bacilli, the child having already become sensitised to tuberculin.

The treatment of epituberculosis should be directed to improving the general resistance by means of prolonged rest and fresh air, sunshine, and a nourishing diet.

Tuberculous Pleurisy

It is not often that a dry pleurisy can be detected during the course of intra-thoracic tuberculosis, although that it occurs is amply proved by the presence of pleural adhesions at post-mortem examination. The most common form of tuberculous pleurisy to be diagnosed during life is pleurisy with serous effusion. If the fluid in these cases be examined it will be found to be clear, straw-coloured, with a lymphocytic cell reaction. The fluid is sterile on culture, but may give rise to tuberculosis when injected into a guinea-pig.

Tuberculous pleurisy with effusion tends to occur at a slightly higher age than the other forms of intra-thoracic tuberculosis. In 19 consecutive cases under twelve years of age the youngest was three and a half, and the average age was six and a half years.

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[1] Eliasberg, H., and Newland, *Jahrb. f. Kinderhk.*, Berlin, 1920, **93**, 88.

[2] Oppenheimer, E. H., *Bull. Johns Hopkins Hosp.*, 1935, **57**, 247.

The onset is usually acute, with pain in the side, and a temperature of three or four degrees, the fever gradually settling in two or three weeks. In other instances the onset is more insidious, the child being brought for advice on account of increasing breathlessness and cough, and on examination the signs of pleural effusion are discovered.

The physical signs are the same as in adults. The affected side of the chest shows impaired movement, the percussion note is dull, breath sounds are absent or may be distantly tubular, and voice sounds are absent. A boxy or tympanitic percussion note may be obtained above the level of the effusion. The heart and mediastinum are displaced towards the opposite side of the chest, and Grocco's triangle of dullness to percussion may be found at the base of the sound lung.

Diagnosis. As a rule this presents no difficulty. The most important condition which must be distinguished is empyema. A history of pneumonia at the onset and a leucocyte count above 25,000 with a relative excess of polymorphs are points in favour of empyema. The temperature is raised in both conditions, but on the whole is likely to be higher in serous than in purulent effusions. These are, however, small points of distinction compared with the examination of the fluid obtained on exploratory puncture of the chest.

Prognosis. Generally speaking, the outlook for children with tuberculous pleurisy with effusion is good. With rest in bed the temperature gradually declines and the signs of fluid recede, but six weeks or more may elapse before the effusion is entirely absorbed. Often there is no further evidence of active tuberculosis, but a long period of convalescence should be insisted upon, and periodic examinations should be made to be sure that the intra-thoracic infection is not spreading.

Treatment. The most important aspect of treatment is absolute rest in bed, combined with fresh air. Treatment on a balcony is of great value. So long as the temperature is raised the diet must be light, but later on, eggs, milk, and cod-liver oil should form part of a full nourishing diet.

Mention has been made of the use of the exploring needle in diagnosis. It is seldom wise to aspirate large quantities of the fluid, because by causing collapse of the underlying lung it ensures rest for the diseased part. Fluid should be drained off freely only when the effusion is extensive enough to cause severe dyspnœa or excessive displacement of the heart with embarrassment to the circulation, as indicated by cyanosis or tachycardia.

The child should not be allowed out of bed until the temperature has been normal for a month and all signs of effusion have disappeared. There should then follow a long period of convalescence at the seaside. Drugs play only a minor *rôle*. Opiates may be given in the early stages in order to relieve pain or reduce coughing, and an iron tonic should be given with cod-liver oil and malt during convalescence.

Diagnosis of Intra-thoracic Tuberculosis. With the exception perhaps of tuberculous pleurisy with effusion, a diagnosis of intra-thoracic tuberculosis is probably made more often than is justified. The various physical signs that arise in each variety of tuberculous infection of the chest have already been detailed. A point on which very considerable value must always be placed is a history that the child has been in contact with an adult known to be suffering from pulmonary tuberculosis. When the physical signs are equivocal, clinical evidence of intestinal or peritoneal tuberculosis will point strongly to the pulmonary condition being also tuberculous.

Of conditions that are likely to be mistaken for tuberculosis, delayed resolution of the lung after an attack of broncho-pneumonia is the most common. The loss of weight, irregular fever, and frequent cough, together with signs of partial consolidation with added moist sounds, all of which occur in unresolved broncho-pneumonia, may make diagnosis very uncertain, and particularly when this picture occurs at a short interval after measles or whooping-cough, in fact it may not be until a few months at the seaside have led to recovery that one feels sure of the non-tuberculous nature of the condition. The wasting, sweating, and low fever that accompany a chronic empyema may also be misleading, particularly if the empyema is shut off between the lobes of a lung. Pulmonary fibrosis and some of the milder cases of bronchiectasis may also offer difficulty, but the long history would be a point against tuberculosis.

The most convincing proof of intra-thoracic tuberculosis is the recovery of the bacillus from the sputum. In childhood, however, unless the lung is rapidly breaking down, sputum is not expectorated, but is swallowed, and therefore special measures must be taken to recover the organism. Three methods can be employed. The child may be stimulated to cough or gag by putting a swab to the back of the throat, and the sputum that is then coughed up may be caught on the swab and examined. This is not a very reliable method. A much better method is to wash out the stomach before breakfast, having first encouraged the child to cough. The stomach contents are then centrifugalised and the deposit examined for the organism, or the deposit may be injected into a guinea-pig, although, of course, if that is done some few weeks must elapse before it will be known whether the guinea-pig has been infected. The third method is to examine the fæces for tubercle bacilli. By one of these methods it is often possible to prove a diagnosis of tuberculosis even at a stage when the coughing is slight and the disease is in its early stages. That children with pulmonary tuberculosis do swallow infected sputum has been known for many years, ample proof being furnished by post-mortem examination, for in roughly half the cases that die of intra-thoracic tuberculosis there is evidence of recent tuberculosis in the intestine or mesenteric glands, but this is evidence which applies to advanced and fatal cases, and the value of finding the organism in gastric washings or fæces lies in the fact that positive results can be obtained at an early stage when recovery is still possible.

The employment of tuberculin skin tests has already been mentioned (see p. 248). In the detection of early intrathoracic tuberculosis, that is to say, at a stage when recovery under treatment may be expected, these tests are of great service, and should be employed more widely than is done at the present day. In doubtful cases a negative test is of much value in excluding tuberculosis, but it must be remembered that when tuberculous disease is rapidly advancing or has become generalised (*e.g.* tuberculous broncho-pneumonia, miliary tuberculosis, tuberculous meningitis), the child may lose the power to react, and the test will then also be negative. A positive reaction up to three years of age almost invariably indicates an active lesion, and often does so during the next two years ; but the interpretation of a positive result in older children calls for caution, as after the first five years of life a positive test is as likely to mean that a child has had tuberculosis and has recovered from it as that the present symptoms are tuberculous. The associated symptoms and signs and X-ray changes must be taken into consideration when assessing its importance.

Finally, valuable help is often to be obtained from the X-ray examination, especially in early cases when the disease has perhaps only reached but not extended beyond the mediastinal glands. In such cases a lateral view of the chest is likely to be of as much, if not more, help than a direct antero-posterior view.

Prognosis. Intra-thoracic tuberculosis occurring under the age of three years must always be regarded as a severe illness owing to the risk of general dissemination, but in the absence of this complication, and provided the diagnosis is made early, eventual recovery may be expected. After about five years of age the disease tends to run a slower course, and there is a greater chance of the area of infection becoming localised because the child is better able to build up his powers of resistance. Tuberculous broncho-pneumonia is usually fatal and the acute miliary form invariably so.

Treatment. The principles of treatment are the same for children as for adults. A combination of rest and fresh air are the two essentials. When investigation shows that a child has been infected by a phthisical adult, further contact must of course be strictly prevented.

With regard to rest, the child must at first be nursed in bed all day. The temperature and pulse rate should be recorded daily, and the weight should be taken once a week, for these will be found the best guides as to when the child may be allowed up, and of the amount of exercise he may undertake.[1] Not only must the temperature remain normal, but the pulse rate should also be within the normal limits for the age, and the weight chart should show a slow steady gain. It is as well for the child to remain strictly in bed until the temperature and pulse rate have been normal for a month, and thereafter the period each day for which the child is allowed up must be only gradually lengthened, while the amount of exercise must be well short of the point of fatigue.

Fresh air is of almost as much importance as rest. The child should be nursed in a sunny room with windows open both day and night, and during the day nursing on a balcony is ideal. Direct sunlight is of great benefit when the fever has subsided, but until then its effect must be carefully watched, for it is sometimes exhausting, and may even prolong the fever, and the same is true of artificial-light baths. The air at the seaside is particularly beneficial, and every effort should be made to arrange for the child to be nursed at the coast. Mountain air, for those who can afford to send their children abroad, is also of great value. The exact choice of residence, however, embraces other aspects, for it is by no means an ideal arrangement to send a tuberculous child to a sanatorium which caters for phthisical adults, on account of the possibility of re-infection. Because children so seldom cough up their sputum, the likelihood of a young child infecting other children at an ordinary convalescent home is probably slight, but, nevertheless, is a risk which most homes will not take. There is at the present day a great need of special sanatoria for children. If the parents are not in a position to make their own arrangements, the most satisfactory course once the convalescent stage has been reached is to arrange for the child to be boarded out in a suitable locality with healthy adults.

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[1] The sedimentation rate of the red cells has also proved to be of much assistance in assessing the child's progress. For the technical details of the estimation, the reader is referred to Dr. Payne's paper (*Lancet*, 1932, *i.*, 74). In health the rate of sedimentation should not exceed 10 mm. in one hour, and so long as the sedimentation rate is above this level the infection must be regarded as active. The test should be repeated at fortnightly intervals.

The diet needs to be of simple nourishing fare, and must be kept in proportion to the appetite. There is nothing to be gained by coercing the child to eat more than he wants. When the appetite is poor, some such mixture as the following, given five minutes before meals, will be useful :—

℞ Sodii bicarbonatis gr. v.
Tincturæ nucis vomicæ m. ii.
Infusum gentianæ compositum ad ʒi.

Milk, eggs, butter, and cheese should form a regular part of the diet, and provided that the child will tolerate it a teaspoonful of cod-liver oil and malt may be given after meals. There is also probably some benefit in giving calcium, a suitable preparation being syr. calcii lactophosphatis in teaspoonful doses two or three times a day.

In the early stages if coughing is a troublesome symptom an opiate linctus should be given, such as the following :—

℞ Tincturæ camphoræ compositæ m. x.
Syrupi scillæ m. x.
Syrupi tolutani m. x.
Glycerini m. x.
Aquam ad ʒi.
For a child of two years and upwards.

Older children should be encouraged to spit out any sputum into a cup put at the cot side, instead of swallowing it.

Sweating is seldom sufficient to need treatment, but small doses of belladonna and tepid sponging will lessen this symptom. Surgical measures such as artificial pneumothorax are seldom suitable for children because the adult type of the disease is so infrequently met with, but it should be considered in older children who show a chronic localised type of infection. The technique of artificial pneumothorax presents no greater difficulty than in adults. In the acute miliary or broncho-pneumonic types, treatment can only consist of rest and the alleviation of symptoms.

Prevention. The first and most valuable point is to secure for the child freedom from contact with relatives or others who are known to have pulmonary tuberculosis. The best method, if a parent is known to be infected, is for him or her to be removed from the home to a sanatorium, but if this cannot be managed then the child should be sent away at once to stay either with healthy relatives or at a residential school. The isolation at home of a tuberculous adult from his children is seldom sufficient. The importance of separating children from known sources of infection is far from sufficiently realised in this country. Such separation cannot be enforced ; it can only be brought about by educating public opinion as to the danger incurred when young children are exposed to contact with infected adults.

The diagnosis of tuberculous infection in its incipient stages may also be regarded as a means of preventing the more severe forms of the disease, for so long as the disease is confined to a primary lung focus and involvement of the nearby glands, recovery under treatment may be expected. In this connection attention may be drawn to the importance of phlyctenular conjunctivitis and erythema nodosum, two conditions which very often signify a recent infection with tuberculosis. Their appearance in a child should always prompt a close scrutiny for other evidence of tuberculosis, including not only a careful clinical examination, but also such auxiliary aids as radiography and tuberculin skin tests.

A wide trial has been made in other countries of a method of immunising infants by giving them orally small doses of a vaccine of specially prepared avirulent bacilli, first introduced by Calmette and Guérin (B.C.G. vaccine). Although promising results have been reported, the safety of the vaccine is still under review, and the method must at present be regarded as *sub judice*.

CHAPTER XVII

DISEASES OF THE CIRCULATORY SYSTEM

Introduction

THE study of the circulation in childhood shows certain differences as compared with the adult. During the first year the pulse-rate is in health about 100 per minute, and for the next three or four years the normal rate lies between 80 and 90, while from the second dentition until puberty the rate is generally between 70 and 80. The rate of the heart depends largely upon nervous control, and in children nervous instability in this, as in other directions, is very common. Thus it is by no means unusual for the pulse-rate of a nervous child to increase during examination to as much as 140 or more per minute; as examination proceeds the pulse-rate may steadily fall, at other times a nervous tachycardia is maintained throughout the examination.

The apex beat in children is normally situated just inside the left nipple line, but is often a space higher than in adults, being found in the fourth intercostal space. The heart is, however, not a rigidly fixed organ, and if the child has been lying on the left side for some time the apex beat may be found to have moved well outside the left nipple. On percussion the area of cardiac dullness should extend to the left just short of the nipple line, and to the right about half a finger's breadth beyond the right border of the sternum. The upper border is less easily defined but is roughly in the second intercostal space.

Certain peculiarities in the heart sounds are to be noted. Normally the pulmonary second sound is louder than the aortic second sound until the tenth year, while from then until puberty the sounds are equal; it is not until after puberty that the aortic second sound becomes the louder of the two. In many normal children a third heart sound can sometimes be heard just internal to the apex beat. It occurs during diastole as a short muffled sound not unlike the normal second sound, from which, however, it is too far removed into diastole to be looked upon as a mere reduplication of the second sound. Its cause is uncertain, but it occurs sufficiently often in healthy children to be of no pathological significance.

The blood pressure in children is lower than in adults. The systolic pressure varies from about 70 mm. Hg. soon after birth up to about 110 mm. Hg. at puberty, while the diastolic pressure during this period varies from 50 to 70 mm. Hg. Blood pressure readings in children are more open to technical errors than in adults, and therefore too much importance should not be attached to slight variations from the normal figures.

A question which often arises is whether slight modifications in the physical signs of the heart are to be regarded as evidence of heart disease. This is sometimes a matter of considerable difficulty, but on the whole the tendency is to regard too seriously what is really no more than a variant of the normal. The problem so frequently arises and is so important that it is not out of place to consider some of the symptoms and signs bearing on this question.

To take symptoms first, fainting attacks are quite frequent during the school years in rapidly growing children, and often arouse parental fears of heart disease. Actually, however, fainting is a very unusual symptom of organic heart disease in children, in fact so uncommon that a complaint simply of fainting is almost an assurance that the heart is unaffected. The reason for this is that fainting due to cardiac disease is generally the result of pure aortic regurgitation, and in children acquired aortic lesions almost always arise secondarily to rheumatic mitral disease, and what symptoms there are will be due to the mitral lesion. In adults the two common causes of pure aortic regurgitation are syphilis and atheroma, but in children the former practically never affects the heart, while the latter does not occur in the young, at any rate to a degree sufficient to cause symptoms. Complaints of lassitude, rapid fatigue, and præcordial pain may or may not be accompanied by organic heart disease, and one must be guided by a careful clinical examination. Præcordial pain occurs in pericarditis and cardiac failure, but is otherwise seldom due to heart disease, although just occasionally acute pain of an anginal character may occur when there is a large aortic regurgitation, and in the more severe forms of congenital heart disease. One of the most important symptoms of organic heart disease is breathlessness, and in severe cases is likely to be combined with a degree of cyanosis. Lastly in a doubtful case considerable importance must attach to a previous history of rheumatic fever, since rheumatism is in childhood the most common cause of acquired heart disease.

Turning now to physical signs, tachycardia, when due to organic disease, is as a rule associated with an increase in the area of cardiac dullness, or with a soft and muffled quality to the first sound, indicative of a weak muscle; alternatively tachycardia may be nervous in origin. When doubt exists help can be had from a record of the pulse-rate taken while the child is asleep. Normally the sleeping pulse is about ten beats slower per minute than the waking pulse ; in nervous tachycardia the day pulse is raised but the sleeping pulse-rate is normal, while if the heart is actively inflamed the sleeping and waking pulse-rates approximate to each other, and are both raised above the normal.

Difficulty is sometimes experienced in deciding whether a cardiac bruit is to be taken as meaning heart disease. It should be recalled that " functional " bruits (that is to say bruits not due to organic disease of the heart) are only to be heard during systole, and therefore if a diastolic murmur is audible as well as one in systole there can be no doubt that organic disease is present. A functional systolic murmur is most commonly heard over the pulmonary area, but may occur over other valves ; when situated at the apex the range of conduction is usually small. The occurrence of a thrill almost always points to organic heart disease. The effect of posture on a systolic murmur should also be noted, for a functional bruit may be more easily heard when the child is erect, while one due to organic disease is either unaffected by posture, or may be more easily heard when the child is lying down.

One special variety of functional bruit is the cardio-respiratory systolic murmur. This is generally localised to the region of the apex beat, and its intensity varies very greatly between inspiration and expiration, in fact it may disappear altogether during a deep inspiration. Its recognition is important inasmuch as it has no pathological significance.

Another common source of difficulty is the interpretation to be put upon a short first sound at the apex, or on reduplication of the heart sounds. A short first sound accompanies tachycardia from any cause, but in nervous tachycardia it has a thumping quality, while if the rapidity of the heart is due to weakness of the muscle the first sound is both short and soft. Reduplication of the heart sounds *without other evidence of heart disease* may be safely disregarded, but a double second sound is sometimes to be heard at the apex accompanying a rheumatic mitral systolic murmur.

ABNORMALITIES OF RHYTHM

Sinus Arrythmia

This form of irregularity, which consists of a speeding up of the heart rate during inspiration and a slowing during expiration, is quite common in childhood and has no pathological significance, indeed it is only likely to be heard when the heart is perfectly healthy, and disappears if for any reason the heart muscle becomes damaged. It may be met with in well-compensated rheumatic valvular disease, but is not present when the rheumatic inflammation is active. As in adults, sinus arrythmia becomes more noticeable on deep breathing.

Extra-Systoles

These can as a rule be easily recognised, for they consist of an extra heart beat pushed into the normal cardiac rhythm, the interval between an extra systole and the preceding contraction being shorter than normal, while a longer pause than usual occurs before the next normal beat. They vary considerably in frequency but may occur as often as once in every three or four beats, and occasionally—as for instance in over-dosage with digitalis—an extra-systole may occur after each normal beat.

Extra-systoles are caused by a contraction of the heart starting from some abnormal situation in one or other of the four chambers, and the localisation of this abnormal focus can be determined by an electrocardiogram. They do not occur in children who have absolutely normal hearts, and their presence indicates that at some time or other the heart muscle has been injured by disease. They may be found in rheumatic carditis after the acute phase has settled down, and they are not uncommonly met with after the infectious fevers, particularly influenza and diphtheria. As a rule they are not associated with other clinical evidence of heart disease, and they do not of themselves give rise to symptoms. They often disappear during exercise, to return again when the heart is beating slowly. With prolonged rest in bed they become less frequent but they do not entirely disappear, and generally as soon as the child gets up and about their frequency increases again. Neither the immediate nor the remote prognosis seems to be in any way affected by the occurrence of extra-systoles, and as they are not abolished even after long periods of rest they do not call for treatment.

Paroxysmal Tachycardia

This is probably rare in childhood, although it is a condition which is easily overlooked, for unless a child is actually examined during an attack there may be nothing to indicate its occurrence. It may be met with at all ages, and an instance has been recorded by Worley in an infant only four days old.

Paroxysmal tachycardia is due to the presence of some irritable focus in the myocardium, which, during an attack, sends out a rapid succession of impulses causing contractions of the heart in place of those normally originating from the sino-auricular node. The abnormal focus may be associated with congenital deformity of the heart or with acquired myocardial disease.

Symptoms. Each attack begins suddenly, the heart rate rising to about 200 per minute or even higher, and after lasting for a variable time, which may be minutes, hours, or days, ends just as suddenly as it began. Shookoff recorded an instance in a child which went on for twenty-four days, but such is quite exceptional. During an attack the child may complain of discomfort or actual pain in the chest, and may rapidly become cyanosed, breathless, and exhausted. Vomiting is common, and giddiness, fainting, or epileptiform fits may occur.

Diagnosis. The diagnosis can only be made if the child is seen actually during an attack, when the turbulent and uncountable heart action will suggest the true state of affairs. Short attacks which last only a few seconds and are accompanied perhaps by vomiting or fainting or cyanosis may be mistaken either for an epileptic fit or for a simple attack of colic.

Treatment. Attacks often arise for no apparent reason, and therefore their prevention may be impossible, but a carefully regulated life to avoid fatigue or excitement is desirable, and the common disorders of health such as simple infections, indigestion, and constipation, must be prevented. If attacks are recurring frequently full doses of bromide should be given, or should an attack be sufficiently prolonged for symptoms of cardiac failure to appear, an intravenous injection of strophanthin (gr. $\frac{1}{300}$ to $\frac{1}{150}$) may be given, followed by digitalin (gr. $\frac{1}{600}$ to $\frac{1}{240}$) administered orally. A prolonged attack in an infant aged two weeks under the author's care was successfully treated by one dose of strophanthin gr. $\frac{1}{800}$ given intravenously.

Auricular Fibrillation

In children auricular fibrillation is only likely to be met with as a complication of long standing rheumatic heart disease which has progressed to mitral stenosis and decompensation, and therefore only occurs as puberty is approached. Even then it is rare. The symptoms and treatment are the same as in adults.

Heart-Block

Heart-block in children occurs in two forms. One is associated with congenital malformation of the heart, and the other accompanies acquired heart disease.

Congenital heart-block. In this form the block is generally complete, that is to say, the contractions of the auricles and ventricles are entirely independent of each other. The ventricle sets its own pace, and in children this is a little quicker than in adults. Thus in three cases observed by the writer the rate of the ventricle averaged 44 per minute, and even with severe exercise and during fever the rate only rose to 56 per minute. Although the block is present from birth it may remain undiagnosed for several years, because the symptoms seldom amount to more than slight breathlessness on severe exertion and an unwillingness to join in vigorous games. Growth proceeds normally.

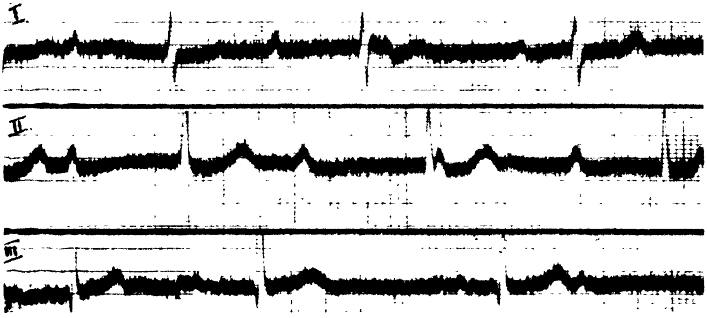

FIG. 59. Electrocardiogram of congenital complete heart-block. From a child aged five years.

Congenital heart-block is usually due to a patency in the inter-ventricular septum which interferes with the continuity of the Bundle of His, and therefore on examination a systolic murmur may be audible over the middle of the heart. In addition a soft bruit can sometimes be heard during diastole, and is probably caused by the contraction of the auricles.

Acquired heart-block. This form of heart-block may be either complete or incomplete, and is due to inflammatory or degenerative changes in the Bundle of His. The complete variety is most commonly the result of diphtheria, while rheumatic disease is the usual cause of incomplete block. In this type severe syncopal attacks may arise, accompanied perhaps by epileptiform seizures, and the condition may even prove fatal. While the congenital variety persists throughout life, the acquired form may be only temporary, passing off completely as the myocardium slowly recovers.

Treatment. In the congenital type the child's life must be so regulated that the amount of exercise keeps within the bounds of cardiac tolerance and breathlessness does not occur, but rest in bed is not called for.

In the acquired variety prolonged rest in bed is necessary in order to give the myocardium every chance of recovery, and the child should not be allowed up until all signs of active inflammation have disappeared. Convalescence must be gradual, and the amount of exercise cautiously increased. Should syncopal attacks occur during the acute stage, they are best dealt with by a hypodermic injection of three to five minims of adrenalin hydrochloride (1 in 1,000).

CONGENITAL HEART DISEASE

Congenital heart disease takes a great variety of forms, ranging from those that are incompatible with life to others which are practically symptomless. As a rule the deformity is due to an arrest of the normal development of some portion of the heart, but certain lesions, such as the valvular atresias and some instances of congenital mitral stenosis and pulmonary stenosis, are to be attributed to infection acquired during intra-uterine life. Another feature of congenital heart disease is its frequent association with other developmental anomalies—according to Still this association is present in 13 per cent.

Symptoms. The symptoms naturally vary very much according to the nature of the deformity. Cyanosis is the most obvious one, but as it does not occur in more than one third of the cases its absence in no way invalidates the diagnosis. Even when cyanosis is present it varies considerably in degree. In certain deformities such as septal defects and patent ductus arteriosus, the shunt of blood is normally from the left to the right side of the heart, and the child does not appear cyanosed ; but under the stress of pulmonary infections or of severe exercise the pressure in the right side of the heart may rise sufficiently to reverse the shunt of blood, and cyanosis will then appear (*cyanose tardive*). With other deformities such as pulmonary stenosis, cyanosis is constantly present, and in the severest cases the livid hue is most striking. The cyanosis is more marked on the extremities and on the exposed parts, the conjunctiva may show a network of purplish engorged veins, and the buccal mucosa may appear plum-coloured. Invariably the extremities feel cold and flabby.

Occasionally cyanosis is present from birth, and survival beyond infancy is then not to be expected. More usually it comes on after some months, or may not appear for two or three years, but once it has appeared it tends slowly to become more profound. The cyanosis is accompanied by an increase in the red cell count, usually in the neighbourhood of 7,000,000 per c.mm., but figures as high as 9,000,000 or 10,000,000 may be reached, while the hæmoglobin is also increased to about 110 to 120 per cent.

Clubbing of the fingers is likely to be present when there is cyanosis, and the degree of clubbing is roughly proportionate to the degree of cyanosis. Clubbing is seldom in evidence until several months after birth, and in its early stage all that may be seen is a heaping up and shininess of the skin just behind the nails. Gradually the nails become broad and curved, and eventually the ends of the fingers may become splayed out and bulbous like a drumstick. Generally the swelling is confined to the soft parts of the fingers but in cases of long standing the phalanges may become broadened from the formation of new periosteal bone.

Of other symptoms, shortness of breath is common. Its severity varies in proportion to the degree of cyanosis, and it is sometimes sufficient to interfere considerably with the child's activities. Sudden attacks of breathlessness may occur, and may lead to syncope or convulsions. Attacks of this sort are most common in infancy, the story being that the child has sudden blue turns in which he pants and then becomes unconscious and may begin to twitch, and when he is seen between attacks there is likely to be a livid tinge to the lips and fingers sufficient to prompt a careful examination of the heart. Other attacks sometimes occur in which the infant becomes pallid and collapsed, and these are often brought on when the child is bathed. Such attacks are undoubtedly dangerous, and when there is a history of them the ordinary bath must be discontinued and the infant should instead be cleansed with warm olive oil. There can be no doubt that in some infants these attacks are associated with a good deal of pain, as may be judged from the short distressed cry which accompanies them, and older children may give a clear account of pain of an anginal character radiating from the chest down the left arm.

Cough is another frequent symptom, and the chest often shows signs of bronchitis, to which children with congenital heart disease are particularly prone. This is probably due in part to an insufficient blood supply in the lungs, and in part to an interference with expansion owing to pressure by the enlarged heart, for collapse of the bases of the lungs is commonly found at autopsy.

Finally mention must be made of the interference with nutrition which congenital heart disease induces. As one would expect, it is most pronounced when there is much cyanosis, but it may also manifest itself quite early in life, in fact it has been pointed out in an earlier chapter that when an infant fails to thrive in spite of an adequate diet the possibility of congenital heart disease must be borne in mind. Not only may the physical growth be impaired, but, in the more severe grades, mental development is also likely to be retarded.

Physical Signs. A few varieties of congenital deformity of the heart can be diagnosed with some degree of assurance by the physical signs, but as a rule it is only possible to take the diagnosis as far as congenital morbus cordis. A point of some practical importance is that the examination of an infant for congenital heart disease may be quite useless unless the child is quiet, and it is therefore wise to examine while a feed is being given.

Inspection of the chest may show nothing abnormal, but in a young infant the sternum and costal cartilages are sufficiently soft to bulge forwards if the heart is much hypertrophied. In infants also the character of the breathing is often peculiar, for with an enlarged heart there is likely to be collapse of the bases of the lungs, which may account for considerable sucking-in of the lateral chest wall during inspiration. Light percussion will confirm the increase of the size of the heart. On palpation a thrill can often be felt ; it is almost always systolic in time, and is accompanied by a murmur which reaches a maximum intensity over the area of the thrill. The presence of a thrill has a diagnostic value in distinguishing a murmur of congenital heart disease from one of functional origin, but it has but little bearing on prognosis, and may be found when there is neither cyanosis nor finger-clubbing.

Almost always a murmur is to be heard, although exceptionally there may be no bruit even when a remarkable degree of cyanosis and gross finger-clubbing leave no doubt of the correct diagnosis. In the great majority of cases the bruit is systolic in time, and has often a noticeably rough quality. Most commonly the murmur is situated over the middle of the præcordia, but it may be over the base of the heart just to the left of the sternum, or towards the apex. Its point of maximum intensity must always be carefully determined, for systolic murmurs due to *acquired* heart disease are loudest over the surface marking of one or other valve, and if this is found not to be the case the probability is that the lesion is congenital. The extent to which the murmur is conducted depends partly on its loudness and partly on the nature of the lesion; thus it may be localised to a small area, or may be heard over the whole chest both back and front. Whether or not a diastolic murmur is present depends on the nature of the deformity, for instance it is sometimes to be heard when there is a patency in the inter-ventricular septum, and is usually present when the ductus arteriosus is patent.

Although an X-ray of the chest is seldom necessary in making a diagnosis of congenital heart disease, the film is often instructive. When there is a patency of the septum or pulmonary stenosis the two ventricles may be practically equal in size and the outline of the heart may then be more globular than normal. When the ductus arteriosus remains open a bulging can sometimes be seen at the base of the heart on the left side.

Diagnosis. One of the most helpful points in making the diagnosis of congenital morbus cordis is the age at which the condition of the heart was first noticed. Rheumatic heart disease, which is the only other common organic form of heart disease in children, is so exceptional under three years of age that a bruit discovered before this age is almost certainly due to a congenital defect. The character of the physical signs, together possibly with cyanosis and finger-clubbing, generally leaves no doubt of the condition. Persistent cyanosis from causes other than heart disease is rare in childhood; both cyanosis and finger-clubbing may occur in bronchiectasis, but they do not reach the same extent, and in addition the history and the physical signs make the differentiation easy; exceptionally a cyanotic tinge may be associated with chronic constipation, but the cyanosis is then unaccompanied by finger-clubbing, while the history of constipation and the absence of any other cardiac signs should prevent error.

The determination of the actual lesion in congenital heart disease is often impossible, but fortunately this is seldom of practical importance because the outlook depends more on the degree of crippling to which the deformity gives rise than on the precise nature of the malformation. There are, however, certain types of deformity which have distinctive signs.

Pulmonary Stenosis

This may be diagnosed in the presence of a systolic bruit localised over the second left intercostal space close to the sternum, and with a short range of conduction. It is generally accompanied by a thrill, and as a rule the pulmonary second sound is softened. Cyanosis and finger-clubbing are always present.

Patent Inter-ventricular Septum (Maladie de Roger)

In this condition there is a systolic murmur of greatest intensity over the middle of the heart, and there is often a diastolic murmur as well. As a rule a systolic thrill can be felt, but cyanosis and finger-clubbing are more often absent than present.

Pulmonary stenosis or atresia and patent interventricular septum often occur together, and together with a dextroposition of the aorta and hypertrophy of the right ventricle make up the Tetralogy of Fallot. Cyanosis usually appears early and reaches a severe degree, and survival beyond childhood is exceptional.

Patent Ductus Arteriosus

The surface marking of the ductus arteriosus is in the second left intercostal space just beyond the border of the heart. The characteristic bruit is situated in this area and is a continuous rumbling sound heard through both systole and diastole, and is fittingly described as a " cartwheel " murmur. There is usually neither cyanosis nor finger-clubbing, and the amount of interference with the child's activities is often surprisingly slight. It has recently been shown that cure of this deformity can be successfully undertaken by ligation of the ductus.

Subaortic Stenosis

This term implies a fibrous thickening of the endocardium of the left ventricle immediately below the aortic valve. The condition is rare. The physical signs are those of aortic stenosis, and consist of a rough systolic murmur loudest over the aortic area and conducted along the main arteries. The murmur is occasionally loud enough to be heard at a distance of a few inches from the child, and is accompanied by a thrill.

Coarctation of the Aorta

This is a condition in which the lumen of the aorta is narrowed at about the level of the entrance of the ductus arteriosus. It is generally regarded as a rarity, although the writer has come across eight instances in the last seven years. The degree of narrowing varies, and in severe instances the aorta may be almost occluded. The effect of the deformity is to raise the pressure in the proximal part of the aorta and in its main branches, while beyond the constriction arterial pulsation is lost. The physical signs can be worked out on the basis of these changes.

Examination shows some hypertrophy of the left ventricle, and increased pulsation can be seen in the episternal notch and in the carotid and subclavian arteries. A systolic murmur is audible over the base of the heart or at the root of the neck, and is conducted along the carotids and into the axillæ. The pulse at the wrist is strong, and the blood pressure in the arms is raised. Pulsation in the abdominal aorta and in the femoral artery at Scarpa's triangle cannot be made out, although in a normal child pulsation at these points is usually easy to feel. Eventually a collateral circulation connecting the upper and lower parts of the trunk is established, and pulsating vessels may be felt and often seen between the scapulæ, or sometimes on the lateral thoracic wall, and the superficial epigastric artery may be also enlarged and visible. A collateral circulation does not develop until about six or seven years of age, but before then the diagnosis can be made with assurance in the presence of the other physical signs enumerated above.

Coarctation of the aorta does not necessarily shorten life, and indeed has been found at post-mortem examination of a man aged 92. There is, however, a risk of ulcerative aortitis developing just beyond the site of the constriction.

Transposition of the Heart

This is a rare condition in which the heart lies in the right half of the chest instead of the left, with the apex beat just within the right nipple line. As a rule the other viscera are transposed as well, and the liver can be felt just below the left costal margin, while the spleen is on the right side. There are usually no symptoms, and the condition is only discovered during routine examination. When the other viscera are transposed as well as the heart the diagnosis can be made without difficulty, and in doubtful cases the characteristic inversion of the deflections in Lead I of the electrocardiogram will settle the matter.

Prognosis. As a general rule the prognosis of congenital heart disease must be judged on the symptoms rather than on the physical signs. When cyanosis appears in early infancy survival into later childhood is unlikely, while in older children if cyanosis is persistent and is accompanied by finger-clubbing, it is unlikely that puberty will be reached. Sudden attacks of breathlessness or syncope in young children are always grave, while in general terms the greater the ease with which dyspnœa is induced the worse the outlook. Another indication may be obtained from the general nutrition and growth of the child, for if this is interfered with to an appreciable extent the prognosis is correspondingly bad. Children with congenital heart disease are more than normally prone to respiratory infections, and attacks of bronchitis or bronchopneumonia may quickly prove fatal. The presence of a congenital deformity of the heart also increases the chance of infective endocarditis. On the other hand the loudness of the murmurs and the presence of a thrill have little bearing on the prognosis.

Treatment. There is, of course, no curative treatment, but careful management may do much to prolong life. Particular attention must be paid to the ordinary rules of hygiene, and a regular action of the bowels and simple dieting are important points. Many of these children feel the cold, and therefore warm gloves and long woollen stockings should be worn.

Drugs are of little importance. Cod-liver oil and malt is of some value, but attempts to build up these children by various extra foods are both unwise and useless, for the very presence of dwarfing indicates that the heart is unable to support a normal weight.

The choice of an ordinary school as opposed to a special school for Physically-Defective children, and the amount of physical exercise that can be allowed, must be judged for each individual child. Extra care should also be taken to protect these children in cold and damp weather, and particularly to shield them from others with coughs and upper respiratory infections.

RHEUMATIC HEART DISEASE

Introduction. Rheumatic heart disease is one of the most serious of all the diseases of childhood, partly because of its frequency but even more so because of its tendency to inflict years of crippling and suffering on its victims. It is by far the most common cause of acquired heart disease in early life.

The term " rheumatism " is used loosely to cover so great a variety of symptoms in adults and children, such as lumbago, sciatica, fibrositis, neuralgia, and the innumerable aches and pains of childhood, that one may wonder whether the word has any meaning. There are, however, three clinical conditions in children which have so close etiological and clinical affinities to each other that there is an advantage in using a term to embrace all three. They are rheumatic heart disease, rheumatic fever (acute polyarthritis), and chorea. The expression "juvenile rheumatism" can usefully be employed to include all three.

It is now realised that all the structures of the heart are likely to be attacked in rheumatic heart disease, and therefore to describe pericarditis, myocarditis, and endocarditis as separate entities conveys a false impression of the disease, indeed it is very doubtful whether rheumatism ever affects the endocardium or pericardium without at the same time attacking the heart muscle, and so the wider term "rheumatic carditis" will be used here.

Etiology. *Distribution.* Rheumatic carditis is widespread throughout the temperate zones, but is rarely encountered in the tropics. Attempts have been made to show that certain physical types are particularly likely to be affected, and red-headed children have been picked out as especially susceptible, but there is no convincing evidence that this is so. There is little doubt that the disease is more frequently found in the densely populated cities and industrial towns than in country districts, and it is likely that such factors as overcrowding, dietetic insufficiencies, and dampness each play their part, particularly overcrowding. Although children of the well-to-do are occasionally affected, rheumatic carditis is essentially a disease of the poorer classes, and in this connection it is of some interest that when children of the poor are reared from an early age in residential institutions, and thereby escape many of the disadvantages of poverty, they very seldom acquire rheumatic heart disease.

The climate, the type of soil, and peculiarities of diet, have each in their turn been thought to have a bearing on juvenile rheumatism, but no direct relationship has ever been established. If the disease is the result of infection, as is now widely held, then it is likely that a generous, well-balanced diet will aid the child's resistance against infections in general, and therefore against rheumatism, but there is no reason to suppose that diet has any closer relation than this.

Heredity. It has long been recognised that several members of a family may be affected by rheumatic carditis, as may also successive generations, and consequently an inherited tendency to the disease has been suggested. Juvenile rheumatism is, however, such a commonplace, and adverse environmental factors are so likely to affect successive generations in the class of population which furnishes the majority of the cases that it is difficult to assign any definite part to heredity.

Age. The age incidence is one of the most striking features

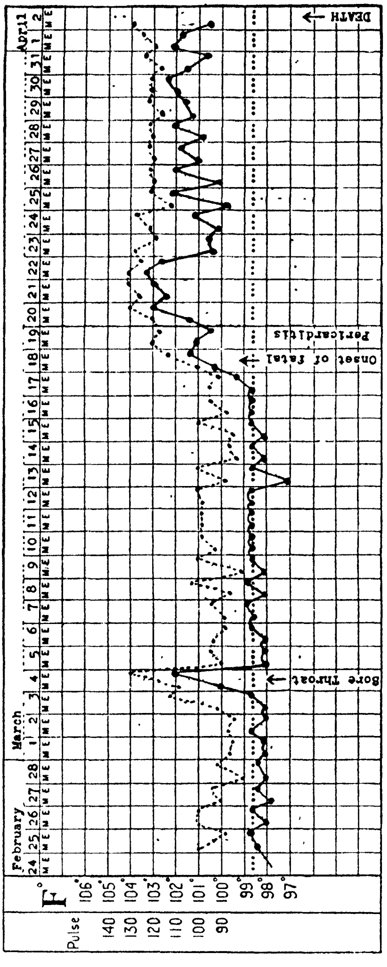

FIG. 60. Temperature and pulse chart of a boy aged ten years with rheumatic mitral and aortic disease, showing a fatal relapse of pericarditis coming on at an interval of a fortnight after a sore throat.

of rheumatic carditis, as it is of the other forms of juvenile rheumatism. The disease is so rare under three years of age that one would properly hesitate before making the diagnosis, and it remains uncommon until the fifth year. The incidence then rises steeply to reach a maximum between the eighth and tenth years, the number of first attacks falling off rapidly as puberty is approached, although recrudescences are often met with during these years.

Before leaving the subject of age, mention must be made of the very rare instances of congenital mitral stenosis which from time to time are recorded. Some are the result of a primary defect in the formation of the valve, others are due to fœtal endocarditis, although whether they are truly rheumatic is uncertain. An instance observed by the author was an infant who died when twenty-three months old at Kings' College Hospital, and autopsy showed a button-hole mitral valve.

Sex. Girls are slightly more often affected than boys.

Relation to Other Diseases. It is well known that the onset of rheumatic heart disease is often coupled with a history of tonsillitis or sore throats. Recent studies have shown that although it occasionally happens that the sore throat and the carditis coincide, much more commonly there is a definite interval of from one to three weeks between the throat infection and the onset of rheumatic symptoms. During the interval the child generally appears well and often returns to school, if indeed he is absent at all, and on this account the actual onset of the heart lesion is likely to be overlooked, so that by the time medical advice is sought the heart may be already seriously damaged. The importance of this sinister period, silent of symptoms, that elapses between the sore throat and the rheumatic condition is insufficiently realised. It is to be emphasised that the infection of the throat has no particular clinical characteristics that make it in any sense " rheumatic ", or that distinguish it from an ordinary sore throat. In some children the throat condition is so mild that it may be entirely overlooked, others may complain of soreness for a day or two but may nevertheless remain at school, although if the temperature were taken it would be found to be raised two or three degrees. Such mild symptoms are particularly likely to appear in children who have had their tonsils removed. If the tonsils are still present the picture may be that of an ordinary follicular tonsillitis, but in others an examination of the throat simply shows a reddened and swollen state of the tonsils and pharynx. The modern conception of the onset of rheumatic heart disease may be said to date from the preliminary sore throat rather than from the actual cardiac manifestations.

It must not be supposed that rheumatic heart disease is a common sequel to sore throat, indeed it is only a complication in probably something under 2 per cent. The factors which determine whether a child will or will not develop rheumatic heart disease after a sore throat are not as yet understood, and it is possible that some change in the child's tissues, of the nature of bacterial hypersensitivity, may be necessary before rheumatic carditis can develop. From time to time small epidemics of rheumatic heart disease have been reported in schools and institutions, so much so that the possible infectiousness of the condition has been questioned. It is likely, however, that these outbreaks depend on a preceding throat infection which has spread by droplet infection from case to case. Regarded in this light one might expect that a wave of tonsillitis in a young community would be followed by a much smaller wave of acute rheumatism, which Glover has shown to be actually the case.

Association with Other Forms of Juvenile Rheumatism. The association between rheumatic heart disease, rheumatic polyarthritis, and chorea, has already been mentioned. Of 226 children with rheumatic heart disease seen at the Rheumatism Clinic at Great Ormond Street, eighty-seven (33 per cent.) gave a history of rheumatic polyarthritis, and fifty-two (20 per cent.) gave a history of chorea. As a rule these other forms of juvenile rheumatism precede or coincide with the onset of the heart infection, and it may be pointed out that even if the heart escapes during the first attack of arthritis or chorea, both these conditions are liable to be repeated and the heart may be involved in a later attack.

It occasionally happens that rheumatic heart disease is discovered quite accidentally during the course of routine examination, in fact this is so in about 14 per cent. of children with carditis. A careful enquiry into the history may fail to bring to light anything to indicate when the onset took place, and even a history of sore throat may be denied, although this is not to say that throat infections have not occurred. Unfortunately the rheumatic process is none the less disabling for having an insidious onset, for it tends to run a slowly progressive course towards mitral stenosis.

Bacteriology. It is now generally accepted that rheumatic heart disease is an infective process, and since the original researches of Poynton and Payne the view that the infection is streptococcal has steadily gained adherents, but the conception of the disease as due to an invasion from the throat or elsewhere of living organisms which settle in the heart and other tissues and there provoke a rheumatic lesion is difficult to correlate with the frequently negative findings on culture of rheumatic lesions, and the general failure to demonstrate a streptococcal blood infection. Claims have been made from time to time for various non-hæmolytic streptococci which have been isolated from rheumatic foci, but without convincing evidence that any of them is a specific cause. Recent studies have shown that when rheumatic heart disease follows a sore throat, the sore throat is as a rule due to hæmolytic streptococcal infection, and it has also been shown that the serum of those suffering from acute rheumatism contains a higher titre of streptococcal anti-hæmolysin and streptococcal anti-fibrolysin than does the serum of normal controls, which indicates that rheumatic children have previously been infected with these organisms. Skin tests performed with the soluble products of disintegrated hæmolytic streptococci also point to the same conclusion. These investigations, however, throw little light on the mechanism which is at work when rheumatic carditis follows a streptococcal sore throat, and an attempt has been made to explain the disease by supposing it to be the outcome of an allergic response of the tissues to streptococcal infection. There can be no doubt that mild streptococcal infections are common enough in childhood for sensitisation to take place, and it is probable that the living conditions which accompany poverty not only increase the likelihood of such infection but also delay recovery, and possibly in this way heighten the state of sensitivity. If the allergic hypothesis is correct, any child who is in this state of hypersensitivity is only waiting for an acute streptococcal infection to set going a train of symptoms which may include one or more of the phenomena of acute rheumatism. It must be pointed out, however, that there is no direct evidence as yet that the histological changes so characteristic of rheumatic inflammation can be brought about in this way.

The possibility that rheumatic carditis may ultimately be found to depend on infection by a filter-passing virus must not be lost sight of. None of the claims in this direction have so far been substantiated, although the recent isolation of virus-like particles from pericardial fluid, and their specific agglutination by the serum of patients suffering from acute rheumatic infection, is of great interest.[1]

Pathology. In childhood the changes observed at post-mortem examination are usually a mixture of the acute and chronic phases of the disease, for it is unusual at this age for death to be simply the result of mechanical failure of the heart from progressive valve changes. The heart is always enlarged, and small sessile vegetations can be seen on the valves, the mitral, aortic, tricuspid, and pulmonary valves being affected in this order of frequency. In addition to recent vegetations, the mitral flaps and chordæ tendineæ, and sometimes the aortic cusps as well, may be thickened and shrunken if there has been a long standing history of heart disease. There is also likely to be evidence of recent pericarditis as shown by swelling and congestion of the pericardium, with a variable amount of fibrinous exudate or fine adhesions, while in others the pericardial space may be obliterated by dense adhesions indicating previous attacks of pericarditis. There may also be inflammatory adhesions between the pericardium and surrounding structures such as the pleura, mediastinum, sternum, or diaphragm. A point of some clinical importance is that there is seldom much free fluid in the pericardium, the great size of the heart in rheumatic pericarditis being due to weakness of the muscle and dilatation of the chambers rather than to any extensive accumulation of exudate.

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[1] Schlesinger, B., Signy, A. B., and Amies, C. R., *Lancet*, 1935, *i.*, 1145.

The characteristic histological features consist of multiple small foci of inflammation situated in the connective tissues in the neighbourhood of small vessels. Each focus, known as an Aschoff nodule, consists of a small central area of necrosis surrounded by an œdematous zone containing plasma cells and lymphocytes, and towards the centre one or more multi-nucleated giant cells. When healing takes place these nodules may either disappear entirely or become converted into scar tissue. Aschoff nodules are most numerous in the myocardium, especially in the wall of the left ventricle at the base of the mitral valve, but may also be present in the pericardium or endocardium, and may be found within the heart valves, producing a valvulitis. Similar nodules are also formed in many other sites of connective tissue, for instance they have been described in the wall of the aorta, and in various of the medium-sized arteries, and in the lungs. The subcutaneous nodules which are to be found over bony prominences, and which, when present, are characteristic of rheumatism, also have the same histological structure.

During the acute stage of the disease there is often a considerable degree of secondary anæmia, and a slight increase of lymphocytes is usual, although insufficient to be of any diagnostic value. Payne[1] and others have shown that the sedimentation rate of the red cells increases with the activity of the disease, and estimations of the sedimentation rate at weekly or fortnightly intervals give a valuable indication of the progress, whether good or bad, which any particular case is making.

Symptoms. One of the most distressing features of rheumatic carditis is the insidiousness of its onset, for it is not at all uncommon for a child to be brought for such vague symptoms as breathlessness, failure to gain weight, or anæmia, and on examination to discover signs of cardiac disease obviously of some considerable duration. On the other hand, if a child is kept under observation after a sore throat, or on account of rheumatic fever, the clinical course of the carditis may be traced from its beginning. In such cases one of the first symptoms is a rapidly increasing pallor, the complexion taking on a waxy or earthy tint.

The temperature at the onset is generally raised to 100° or 101° F. and as a rule remains up for two or three weeks before gradually settling to a normal or subnormal level. In the most severe and fulminating cases, however, the temperature may rapidly fall to 96° F. or so ; such a low temperature makes the outlook very grave, and the temperature is likely to remain at this low level until death takes place. The pulse rate rises with the temperature, and may run as high as 140 or so per minute, remaining raised for several weeks or months so long as active inflammation is present, and settling very slowly some long time after the temperature has returned to normal. Of other early symptoms vomiting and loss of appetite are common. Nose bleeding is not infrequent. Pain over the heart is often complained of, particularly if there is pericarditis ; it is likely to make the child restless and frightened, especially at night-time.

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[1] Payne, W., *Lancet*, 1932, *i.*, 74.

Even if anæmia does not appear at the beginning it becomes obvious after a week or two, and persists long into convalescence. At the beginning the weight often falls rapidly, and the child may soon become wretchedly thin, in fact the loss of weight is sometimes the reason for medical advice being sought. Great value attaches to regular weighings, for the weight offers one of the best guides to the child's progress, and this holds good when the child is under supervision after the active phase is over, for so long as the weight is rising, albeit slowly, it affords a safe indication that no fresh inflammation is going on.

Physical Signs. The earliest physical signs result from a weakening of the heart muscle. The impulse at the apex becomes more diffuse than normal and may extend just beyond the left nipple, and by light percussion the area of cardiac dullness will be found increased. The change in area may be only slight, but in severe cases the dilatation of the heart is both rapid and extensive, so much so that the size of the heart may alter from day to day. On auscultation the first sound at the apex becomes shorter and softer than normal, and this is followed by the development of a soft systolic murmur. It should be noted that the earliest signs of cardiac involvement can be detected before a murmur is heard, and alternatively when a murmur appears a careful examination will enable corroborative signs of mild carditis to be obtained. The mere presence of a systolic bruit localised at the apex, especially if unaccompanied by other signs of cardiac disturbance, is not sufficient evidence on which to diagnose rheumatic heart disease, for in many cases the bruit proves to be temporary, and disappears when the general health of the child improves.

The presence at the apex of a diastolic murmur is a valuable sign of organic disease. The most common murmur in this period of the cardiac cycle is a mid-diastolic bruit. It is separated from the second sound by a short interval, so giving rise to three sounds at the apex—a systolic murmur, then the second sound, followed by a mid-diastolic murmur. Its point of maximum intensity is a little internal to the apex beat, and it is very localised, seldom covering an area of more than an inch or an inch and a half in diameter. The mid-diastolic bruit is probably produced by the ventricle sucking blood from the auricle during diastole through a thickened or stiffening mitral valve.

Observation of rheumatic hearts over long periods shows that the mitral murmurs generally appear in a definite order. The first is a systolic bruit at the apex ; this is followed by reduplication of the second sound at the apex ; the second portion of the reduplicated sound later slurs off into a mid-diastolic murmur. If the disease progresses further the mid-diastolic murmur may be replaced by a short presystolic murmur, which is often interchangeable with a short early diastolic murmur, the presystolic being heard when the heart is rapid, while the early diastolic appears when the heart rate slows. The short presystolic murmur is temporary and is unaccompanied by a thrill, thus differing from the classical rumbling crescendo presystolic murmur of established mitral stenosis.

When the acute inflammation of the heart subsides, the murmurs which have just been described will often be found to retrace their steps. After a few weeks the early diastolic and short presystolic murmurs settle down into a mid-diastolic bruit, which may persist for several months or years. The mid-diastolic murmur may revert still further to a reduplicated second sound, and this may gradually return to a normal second sound. In a few cases the systolic also disappears, and as far as one can detect the heart becomes normal. On the other hand, mitral stenosis may slowly develop from the stage of systolic and mid-diastolic bruits ; the first sound at the apex then becomes gradually louder and the mid-diastolic murmur links up with this loud first sound to produce the rumble and slap typical of established mitral stenosis. Mitral stenosis does not develop for several years after the original attack of carditis, and is therefore not often met with during childhood, but appears at about puberty. Of twenty-nine children with fully developed mitral stenosis the shortest period in which this lesion developed after the first attack of carditis was three years, but more often double that time is required.

The ultimate size of the heart depends largely upon the degree of injury to the valves. When these have been only slightly affected the heart may return to a normal size, but more often the valves remain incompetent, and this has to be compensated by hypertrophy of the muscle, and so the heart remains enlarged. So long as compensation is satisfactorily maintained the heart rate keeps approximately within normal limits, the apex beat becomes strong, and the first sound increases in length. As a rule hypertrophy of the heart does not cause any deformity of the chest wall, but if the first attack of carditis should occur at as young an age as three or four years, at a time when the chest wall is soft and yielding, the sternum and costal cartilages on the left side may develop a definite forward bulge. The likelihood of this is perhaps increased by the fact that when rheumatic heart disease begins before the fifth year it is almost always severe, and, if the acute phase is recovered from, considerable hypertrophy follows.

Although recent vegetations can be found on the aortic cusps in fully half of the cases that come to autopsy, these often form only during the final and fatal attack, for clinical evidence of aortic involvement is only found in 6 per cent. of children with rheumatic heart disease. Almost invariably aortic disease is accompanied by signs of mitral involvement, and as a rule the aortic lesion develops some time after the mitral, perhaps during the second or third attack of carditis. The first evidence that the aortic valve has been affected is the appearance of a diastolic murmur conducted down the left border of the sternum. If the murmur appears during the height of the illness there may be some difficulty in distinguishing it from a pericardial rub, although the scratching superficial character of the latter, and its occurrence generally in both systole and diastole, are points of distinction. It more often happens that the aortic murmur is heard for the first time when the heart is beginning to recover from the acute stage, although the disease must have settled in the valve some weeks before. As a rule once an aortic diastolic bruit appears it is permanent, but this is not invariably so, for instances have been observed in which aortic murmurs, having been in evidence for two or three months, have slowly disappeared. Aortic regurgitation soon leads to hypertrophy of the left ventricle, with displacement of the apex beat downwards into the fifth or sixth intercostal space. A water-hammer quality of the pulse is also characteristic, but it is not found so often or to the same extent in children as in adults.

Pericarditis

Inflammation of the pericardium is likely to develop after other signs of carditis have been present for a few days. The appearance of the child is often the first warning, the expression becomes anxious, the face seems puffy, and the pallor increases. The temperature is usually raised to about 102° F. and the pulse becomes rapid and weak. Vomiting at the onset is usual, indeed sickness and a rapidly increasing pallor in any child ill with rheumatic heart disease always suggests an oncoming pericarditis. Restlessness is often a feature, and may be made worse by a frequent cough. Pain is likely to be complained of, but it is very variable and its severity bears no relation to the amount of audible friction. As a rule it is felt over the heart, but may be referred to the abdomen and so may possibly be confused with some acute abdominal emergency, or it may be referred to the tip of the left shoulder.

Examination shows the apex beat at first diffuse and the heart action turbulent, but as the condition progresses the apex beat gradually fades, owing to the weakness of the heart muscle, until it may be difficult to detect. Percussion shows that the heart is dilated upwards as well as to both left and right. The dilatation may be so great that the heart stretches from the right nipple across to the left axilla, and this is often accompanied by such a damping down of the heart sounds as to suggest the presence of an extensive pericardial effusion, although actually this is seldom the case, the signs being due to great dilatation of the muscle.

The appearance of pericardial frictions puts the diagnosis beyond doubt. They consist of to-and-fro scratching sounds heard at first over the base of the heart, but soon becoming audible all over the præcordia, and with a superficial character as though just under the stethoscope. To begin with they are soft, and can be made to vary with the pressure of the stethoscope, but later on may become so loud as almost to drown the other heart sounds. In the milder cases they may only be heard for a day or two, but more usually last for anything from a week to three weeks, and are last heard near the xiphisternum.

There is no doubt that pericarditis may occur without friction sounds ever being audible, and this can be readily understood from post-mortem examinations, which sometimes show that only a small area of the pericardium, perhaps no larger than a shilling, has been involved, and if this should occur on the posterior aspect of the heart the frictions might well be inaudible.

Not infrequently physical signs can be obtained over the lungs. As a rule they consist of some impairment of the percussion note and diminished or tubular breath-sounds at the lower angle of the left scapula. They are also sometimes present at the base of the right lung. They usually appear within a short time of the pericardial frictions, but are occasionally present a day or two before the frictions can be heard. Post-mortem examination shows that the signs can be produced by a variety of conditions. In some the left lower lobe shows a pneumonic consolidation, with perhaps an associated pleurisy, the lung being a brighter red than usual and very congested. This is the type of lung in which Aschoff nodules may be found, proving the condition to be a true rheumatic pneumonia. In others the lower lobe is collapsed and airless and there may be some free fluid in the pleura. The cause of the collapse is uncertain. It may be due to direct compression by the very large and dilated heart, or else to immobility of the left half of the diaphragm owing to the overlying pericarditis.

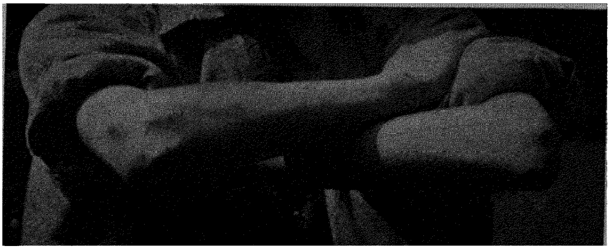

FIG. 61. Rheumatic nodules on the elbows.

Recovery from pericarditis may be so complete that without knowing the history it would be impossible to say that the condition had ever existed, but at other times dense adhesions form, not only between the two layers of the pericardium, but also between the outer layer and the various adjacent structures such as the mediastinum, the pleura, the diaphragm, or the anterior chest wall. When the heart is tethered in this way the clinical condition of *adherent pericardium* results. The effect on the heart is always to cause gross hypertrophy, more than could be accounted for simply by the scarring of the valves. Other signs may also arise owing to the pull of the heart on neighbouring parts ; thus during systole the intercostal spaces over the præcordia may be drawn in, and may be felt to rebound in diastole, while posteriorly the eleventh and twelfth ribs on the left side may also be pulled in during systole (Broadbent's sign). Children with an adherent pericardium are always much crippled in their activities, and succumb to progressive cardiac failure within a few years.

Complications. Rheumatic nodules occur in the subcutaneous tissues in a considerable proportion of cases, and a careful search for them should always be made because they indicate that the inflammatory process in the heart is still active. They are to be looked for over the bony prominences, particularly the olecranon, knuckles, patella, malleoli, on the vertebral spines, along the spines of the scapulæ, and over the occiput. They also occur along the tendons over the dorsum of the feet, and on the back of the hands, and are occasionally present on the tendons crossing the palms, where they may be tender and are likely to cause troublesome flexion of the fingers.

The nodules vary in number from one to a dozen or more, and often develop so quickly that a considerable crop may appear within two or three days. They are as a rule painless, and feel like small firm pellets varying in size from a millet seed to a pea. They attain their largest size over the occiput, where they may be as large as marbles. They remain for a very variable time ; as a rule they disappear after two or three weeks, going as mysteriously as they came, but they sometimes remain for twelve months or even longer, especially over the elbow, and when this happens they gradually become fibrous and may even come to be covered by a small adventitious bursa. By implying that the carditis is still active, their appearance calls for continued rest, although the nodules themselves do not require treatment except when they occur in the palm and cause contractural flexion of the fingers. A light finger-splint should then be worn.

Another occasional accompaniment of carditis is an erythematous skin rash—erythema marginatum—occurring on the trunk and proximal parts of the limbs. It consists of thin, curly, pinkish lines, often appearing as small circles the size of a sixpence. The rash comes up quickly within an hour or so, and it may last from a few hours to several days, during which time its pattern is continually changing. It does not irritate, and is often unaccompanied by fever or other evidence of active rheumatism, although it is very unusual for the rash to appear unless there are also signs that the heart has been involved. It does not call for treatment, and is, in fact, quite unaffected by anti-rheumatic drugs, and is chiefly of importance as indicating that the child is a rheumatic subject. Entirely different is the purpuric eruption on the trunk and limbs which occasionally occurs in the most severe and fulminating forms of rheumatic heart disease.

Pulmonary infarction is an occasional complication, and is indicated clinically by sudden pain in the chest, followed by fever, the expectoration of some blood-stained sputum, and the development of signs of consolidation over a small area of the lung. The embolus causing the infarction probably arises from clots forming in the right auricle, and is to be distinguished from the emboli which occur in ulcerative endocarditis in that it does not cause suppuration. Venous thrombosis is a rare complication, generally affecting the veins of the neck or inferior vena cava, and in one child gangrene of the fingers and toes set in a few weeks before death.

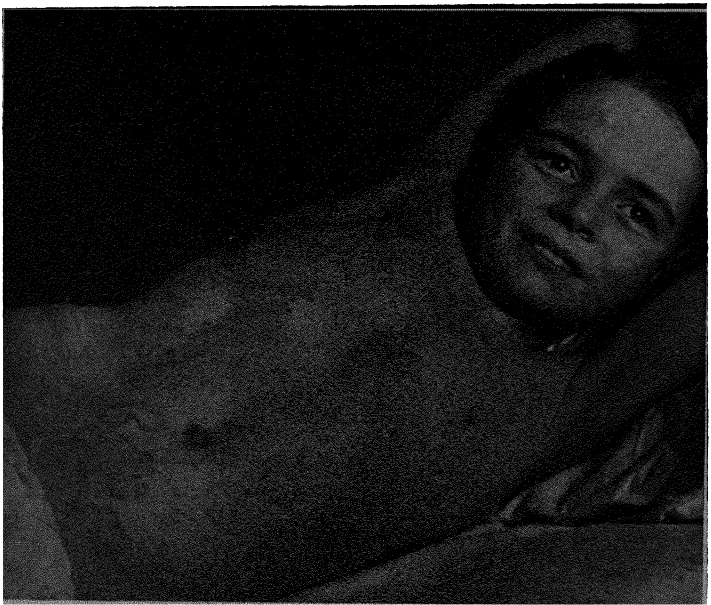

FIG. 62. Erythema marginatum in a boy aged eleven years, suffering from rheumatic carditis.

Diagnosis. As a rule the physical signs of rheumatic heart disease are sufficiently definite for the diagnosis to be reasonably straightforward, but there may sometimes be considerable difficulty in deciding whether, in a child who is otherwise seemingly healthy, a systolic bruit at the apex accompanied possibly by tachycardia is indicative of organic disease. The differentiation between functional and organic bruits has already been considered at the beginning of this chapter. Suffice it here to say that a history of some previous rheumatic condition such as acute polyarthritis or chorea is in itself valuable evidence that the signs in the heart are rheumatic. Of the various physical signs, an increase in the size of the heart is of greater value as indicating organic disease than either tachycardia or the presence of a systolic murmur.

During the acute phase of the disease certain symptoms may be misleading. The pain of pericarditis may be referred to the abdomen, and may give rise to sufficient rigidity of the abdominal wall with superficial tenderness to simulate appendicitis. An error of this sort will be avoided by making a careful examination of the heart. When the pulmonary signs which so often accompany rheumatic pericarditis develop before pericardial frictions become audible, the proper significance of the changes at the left base may be difficult to appreciate. Dilatation of the heart with loud systolic bruits at both apex and base occur in the severe anæmias, and in such cases one may well wonder whether there is not organic disease of the heart, particularly when one remembers the severe degree of anæmia that occurs in acute rheumatic carditis, but the negative history of rheumatism, and the other signs of the anæmic condition such as enlargement of glands or spleen, together with the character of the blood count, serve as distinguishing features.

In acute cases the possibility of ulcerative endocarditis may sometimes arise. Ulcerative endocarditis is rarely seen in childhood, but when it occurs it does not differ from the same disease in adults ; the swinging fever, the presence of an enlarged spleen, the evidence of emboli, and the results of blood culture will help to differentiate such cases. Chronic rheumatic heart disease may at times be difficult to distinguish from congenital heart disease, but the character and position of the bruits in the latter, the possible presence of cyanosis and finger-clubbing, and particularly a history that the heart condition has been present from infancy will help to distinguish them.

Course and Prognosis. Recent studies have shown that the outlook for children with rheumatic carditis is not so uniformly bad as was at one time supposed, indeed a small proportion makes so complete a recovery that all evidence of heart disease vanishes. Of 232 children with heart disease under observation at the Rheumatism Clinic at Great Ormond Street it was found that 10 per cent. had lost all trace of their disease within a period of two years, and in an enquiry into the history of 100 cases of endocarditis after a lapse of ten to thirty years from the initial illness, Morse[1] found that in 37 the heart had returned to normal, 21 showed signs of persistent damage to the heart, and 36 had died. The principal factor in making for recovery is an early diagnosis, for if complete rest and good nursing are at once instituted the heart may be saved from irreparable damage. The importance in this respect of keeping a child under observation for three or four weeks after a sore throat cannot be doubted, and, if the heart has been affected and has recovered, the parents should be warned not to regard lightly a recurrence of throat infection, since the whole sequence of events may be repeated. The fact that children are capable of overcoming rheumatic carditis should be a continual source of encouragement to those whose work lies in helping them in their struggle towards recovery.

The majority of children survive their first attack of rheumatic carditis, but are left with a varying degree of scarring of the heart muscle and valves. Provided no further attacks ensue they may grow up with nothing worse than a mitral regurgitant bruit and some slight cardiac hypertrophy, not sufficient to interfere to any appreciable extent with their activities or livelihood. In others the initial injury is severe, and as recovery proceeds the mitral valve becomes more and more thickened and contracted until the clinical picture of mitral stenosis slowly develops, exerting a progressively incapacitating influence and eventually ending in cardiac failure. Once a heart has been affected there is throughout childhood a risk of recrudescences of acute inflammation, and each such attack means a further permanent injury to the heart, if indeed it does not prove fatal. In yet other cases the child

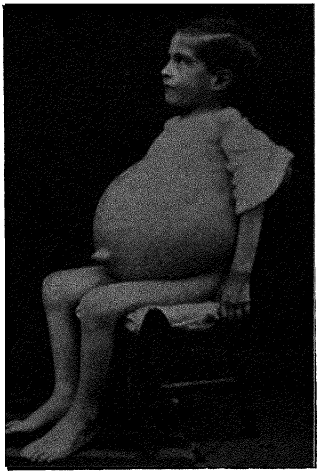

FIG. 63. Cardiac ascites in a boy aged six years, suffering from mitral stenosis (see text).

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[1] Morse, J. L., *Amer. Jour. Dis. Child.*, 1931, **42**, 735.

never seems to make a proper recovery from the initial attack, but the disease slowly smoulders on with an occasional exacerbation every few weeks or months. Such children do badly, and, after perhaps months or a year or two of a steadily downhill course, die during an acute relapse.

In childhood death from rheumatic carditis almost always occurs during or soon after a period of fresh infection. A fatal issue from slowly progressive cardiac failure, the result of sclerosis of the valves, is generally delayed until young adult life, but if the initial illness occurs early enough this sequence of events may be met with towards puberty. An exceptional instance was that of a boy under the writer's care, in whom mitral disease had been present since one year of age, and who had suffered from chronic cardiac failure with such a degree of ascites that the abdomen required tapping on seven occasions before he died at the age of six years (see illustration).

Death from rheumatic carditis is particularly distressing in children because consciousness is maintained to the end, and in the last few days they seem to be aware of their fate, and become restless and frightened. In consequence of this their behaviour often undergoes a peculiar change, for instance they may select one particular nurse who must do everything for them, and they will not suffer anyone else near them, not even their parents. The doctor is, however, exempt from their intolerance because he is thought to possess the power to cure. The only drug that is of the slightest benefit in these cases is morphia, and there need be no hesitation in using it. To a child of ten years $\frac{1}{16}$ gr. of morphia, or $\frac{1}{12}$ gr. of heroin, can be given once or twice a day according to the child's needs. The outlook when this stage is reached is usually hopeless, but apart from the humane desire to allay the child's fears, if a recovery is to be made the anxious restlessness must certainly be checked, and for this purpose the milder sedatives are without effect.

Treatment. This may be divided into four phases :—

The Acute Phase. During the period of active inflammation the two essentials consist of rest and expert nursing. There is no specific serum or drug which will abort an active carditis, and medicines are chiefly concerned with the relief of special symptoms.

Rest must be absolute. The children should be nursed in bed between blankets, lying as flat as is compatible with comfort. Generally they can lie quite flat with one pillow, but if there has been previous damage to the heart this position may perhaps cause breathlessness, and then they will be more comfortable if propped up with several pillows. They should not be allowed to do anything for themselves, but should be hand-fed, and of course must use bed utensils. Treatment carried out by the parents is unsatisfactory as it is practically impossible to get real and adequate rest for the child, and it is particularly difficult for them to realise that the rest must be continued even when the child is beginning to improve. Trained nurses should therefore be employed.

The diet in an illness which runs so long a course needs to be both nourishing and easily digestible. The appetite must be studied, and no attempt should be made to force food on the child. Meals should be small but frequent. To begin with, milk, milk-puddings, broths, jellies, eggs, fish, stewed fruit, and thin bread and butter, will form the main part of the diet.

Adequate sleep is most important, and, if necessary, drugs should be given to secure this. Aspirin or bromide may be sufficient, but when the child is very restless, Dover's Powder (2 gr.) is more suitable.

Pain over the heart is best dealt with by warm poultices such as antiphlogistine or linseed. The use of ice bags suspended over the heart has largely dropped out of fashion. It has been claimed that the cold allays the turbulence of the heart, but children who have had both warm and cold applications prefer the former. Another troublesome symptom is cough, and it should be checked because it adds needlessly to the work of the heart. It is best dealt with by a linctus containing 10 or 15 minims of tinct. camph. co. Vomiting is another symptom which may call for treatment. The ordinary gastric sedatives are often without effect, but a minim of tincture of iodine in a wineglass of milk may be successful, and can be combined with warm fomentations over the epigastrium.

The use of sodium salicylate and aspirin calls for comment. Both have had an extensive trial owing to their value in rheumatic arthritis, but whether they influence the course of carditis is doubtful. Their most obvious effect is to bring the temperature down to normal or even to subnormal, but the pulse rate does not drop correspondingly, and so long as there is active inflammation the children often appear more comfortable if the temperature is raised one or two degrees than when it is artificially depressed. These drugs are chiefly of service in alleviating any pain in the joints and limbs which so often accompany carditis. Their use after tonsillitis as a prophylactic against relapses of carditis is mentioned later. It may be added that the sulphanilamides appear to be without effect in rheumatic carditis.

With regard to cardiac stimulants, considerable caution is needed. Whatever is the theoretical view of brandy, children often seem better while receiving it, and it is sometimes helpful in checking vomiting. A teaspoonful may be given in an ounce of water two or three times a day. Strychnine has seemed of value when the heart is dilating rapidly ; three or four drops of the tincture of nux vomica may then be given with syrup to disguise its bitter taste.

Care must be exercised in using digitalis. As a general rule it should not be given during the stage of acute inflammation, for although it may cause a fall in the pulse rate the extra effort required at each beat may place too great a strain on the muscle, and after a few days the heart may quickly dilate and fail. On the other hand when an acute relapse of carditis is superimposed on an old cardiac lesion for which digitalis has already become necessary the drug should be continued. Its use in slowly progressive cardiac failure is dealt with later. The beneficial effect of digitalis in certain cases during recovery has been pointed out by Sutherland. It sometimes happens that several weeks after the onset of acute carditis the pulse remains rapid, although to judge from the child's colour, his appetite, his increasing weight, and the diminution in the size of the heart, he seems to be definitely on the mend, in fact the persistent tachycardia is the only bar to allowing the child to sit up out of bed. Digitalis may then be given with advantage, and should be continued until the pulse has fallen to eighty or ninety beats per minute. If the tincture is used five minims three times a day may be enough, or the dose may be gradually increased. Digitalin granules (Nativelle) gr. $\frac{1}{600}$ are more reliable, and may be given once or twice a day. Whichever preparation is used the occurrence of vomiting, or of an extra-systole after each normal heart beat, indicates that the drug is being used too freely, and it must be omitted for a few days.

Cardiac Failure. The second phase of treatment arises when there is progressive cardiac failure, as indicated by cyanosis, pulmonary congestion, enlargement of the liver, and widespread œdema. Such failure may arise while the heart is actively inflamed, or may be due to a failure of compensation from increasing mitral stenosis. The withdrawal of a small amount of blood is one of the best and quickest ways of giving relief. If there is active inflammation the application of two or three leeches over the præcordia is an equally satisfactory method, as the blood is drawn off slowly and in small amount, but in chronic failure venesection is better since it is necessary to take off an ounce or two of blood. The improvement in the child's comfort after blood-letting is often striking.

The bowels should be kept moving freely, and for this purpose repeated small doses of calomel, such as gr. $\frac{1}{4}$ three times a day, are very useful. As a rule the output of urine falls as the signs of cardiac failure increase, and efforts should then be made to provoke diuresis. Diuretin (gr. $7\frac{1}{2}$ to 10), or theocin sodium acetate (gr. 2) can be given thrice daily with benefit, but if the œdema is at all severe better results are obtained from the intravenous injection of mercurial diuretics such as salyrgan, of which 1 c.c. may be injected once or twice a week. This usually leads to a considerable rise in the output of urine lasting for about forty-eight hours.

Digitalis[1] is also of great value in this stage; it is often a good plan to start with fifteen drops of the tincture every four hours for four doses, and then to drop the dose to $7\frac{1}{2}$ m. three times a day. The optimum dose must be worked out for each individual child, the pulse rate being kept down to about ninety per minute, for if it is kept persistently lower signs of overdosage, such as vomiting or coupling of the heart beats, are likely to appear. Digoxin[2] is a more potent preparation, of which 0·25 mgm. may be given once a day. Tincture of strophanthus (two or three minims ter die), may be used in the place of digitalis, but it is a less easy drug to control.

When the amount of ascites becomes enough to make the child uncomfortable or to embarrass breathing the fluid should be removed by tapping. Œdema of the feet is sometimes severe, and can be dealt with by two or three stab punctures through the distended skin; this is preferable to using Southey's tubes, since drainage is just as efficient, and the risk of sepsis is less. The resistance of the œdematous tissues against infection is feeble, and therefore if drainage is carried out strict aseptic precautions must be taken.

Convalescence. The third phase concerns the management of convalescence, at a time when the acute inflammatory process has subsided and the child is making a gradual return to as normal a life as possible. This stage is a long and difficult one, for it is important that the child should not be allowed to advance too quickly, yet at the same time the encouragement of invalidism by too prolonged rest is equally undesirable. It is sometimes difficult to be sure when this stage should begin. Regular estimations of the sedimentation rate of the red cells at weekly intervals are helpful, for the child should not be allowed up until the sedimentation rate has fallen to normal.[1] Another difficulty sometimes encountered is that the pulse rate, which has been normal so long as the child has been resting in bed, rises twenty or thirty beats per minute every time an effort is made to start getting him up. This may be due to nervousness or excitement, and the best way of telling this is to take the pulse when the child is asleep, for if the rapid day pulse is due to heart strain the sleeping pulse will also be raised, but if the sleeping pulse is down to about 80, the rise in the day pulse can be discounted.

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[1] Nativelle's granules of digitalin (gr. $\frac{1}{600}$ once or twice a day) are less toxic than tincture of digitalis. The writer prefers tab. digoxin 0·25 mgm., once or twice a day, each tablet being the equivalent of fifteen minims of the tincture.

[2] Digoxin (B.W. & Co.) is prepared from the leaves of digitalis lanata instead of digitalis purpurea.

The convalescent stage is most conveniently managed at one of the convalescent homes especially intended for children with rheumatic heart disease,[2] where they can stay for many months, and meanwhile receive a certain amount of education. A system of carefully graded exercises is arranged, starting from complete rest in bed and ending with the child being up all day except for a mid-day rest. The complete course consists of about half a dozen grades, and the progress of the child from one grade to another is checked by the effect on the pulse rate and on the weight. A study of children at these Homes shows that occasionally the heart signs entirely disappear, but more often the aim must be to establish a high degree of compensation for a heart in which signs of valvular incompetence persist. By the time the child returns home questions such as whether he is fit to walk up stairs, to attend school, and to take part in games, should have been decided. When the heart has been severely damaged attendance at a special " Physically Defective " school may be advantageous, if there happens to be one in the district. Otherwise it may be advisable to arrange for half-time attendance at school.

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[1] By Payne's method (*Lancet*, 1932, *i.*, 74) the red cells normally sediment from 3 to 6 mm. in one hour. Anything above 10 mm. should be considered abnormal. In active carditis the figure may rise as high as 60 mm.

[2] In London and the Home Counties special convalescent homes have been established for children with rheumatic heart disease: at Carshalton (L.C.C.) and Brentwood (L.C.C.), at West Wickham and Kilburn (under the Invalid Children's Aid Association), the Children's Heart Home at Lancing, and St. John's Open-Air School for Boys at Chigwell.

During the convalescent period the diet should be as full and nourishing as possible. Drugs in the ordinary sense are not required, but general tonics such as Parrish's Food or equal parts of malt and syrup of iodide of iron may be given with advantage.

Prevention. The fourth phase concerns prevention, for the desirability of this is obvious in a disease which gives rise to so much crippling and in which complete cure is only seldom obtained.

The frequency with which throat infections precede rheumatism raises the question of tonsillectomy as a preventive. It may be said at once that removal of the tonsils with this object is disappointing, indeed out of 428 children with rheumatism at Great Ormond Street, all of whom had had their tonsils removed, 36 per cent. began their rheumatism at some time subsequent to the operation, and it occasionally happens that the onset of severe carditis follows so closely upon tonsillectomy that one is forced to conclude that the operation actually initiated the carditis. The tonsils and adenoids do not comprise the whole of the lymphoid tissue in the pharynx, and sore throats, sufficient to provoke rheumatism, may occur after a thorough enucleation, and therefore tonsillectomy cannot possibly be a guarantee against the later development of rheumatic heart disease. The question of removing the tonsils must be decided in rheumatic, as in other children, according to the degree of impairment of the general health which they are causing.

It might be thought that the interval of one to three weeks which often elapses between a sore throat and subsequent rheumatism should offer an opportunity for prevention, the sore throat acting as a warning of possible rheumatic sequelæ. Much could probably be accomplished if it became customary to keep all children under medical supervision for a month after a sore throat. During this period the heart should be examined once a week for the presence of commencing carditis, for there is little doubt that the degree of damage to the heart would be considerably lessened if the diagnosis of carditis were to be made at its beginning. Supervision of this sort should not interfere with the child's schooling, in fact it could be well undertaken by the School Medical Service. Recent observations at West Wickham Heart Home and at the Cheyne Hospital for Children have suggested that salicylates given at the time of the sore throat and continued for a month afterwards may thwart the attack of rheumatism, or at any rate diminish its severity.

Aspirin is convenient for this purpose, 15 to 30 gr. being given daily, provided that the child is in bed. This is certainly advisable when a sore throat arises in a child who is already suffering from rheumatic heart disease, for the likelihood of a relapse is then very considerable. Such relatively big doses of aspirin are not practicable for children who are up and about, and therefore should not be used for children who, apart from their sore throat, are otherwise healthy, although even for them small doses such as 5 gr. twice a day can be given with advantage.

Finally, the fact that juvenile rheumatism is so much more common among the poorer classes indicates that measures to improve their living conditions should incidentally lead to a reduction in the incidence of rheumatism. The avoidance of overcrowding, and the cultivation of a higher standard of hygiene in such matters as fresh air, diet, and clothing, are steps in this direction, but are ideals to which a nation can only slowly attain.

Bacterial Endocarditis (Ulcerative or Infective Endocarditis)

This is a rare condition in childhood, and is due to the lodgement of bacteria on the cardiac valves and the mural endocardium, where they give rise to the formation of soft friable vegetations from which emboli are easily broken off causing infarctions in distant parts of the body. The majority of cases are due to streptococcal infection, but pneumococci, staphylococci and other organisms are sometimes present instead. The causal organism can often be cultured from the blood.

The course may be either acute or subacute. The acute form arises as a rule as a complication of some severe infection such as pneumonia, empyema, or tonsillitis. The clinical features comprise a high swinging temperature, sweats, rigors, severe anæmia, and as a rule loud cardiac murmurs which may change their character almost from day to day, and are likely to arise from both aortic and mitral valves, and possibly the valves of the right side as well. Emboli are common, and according to their site may account for petechial hæmorrhages in the skin, an enlarged and tender spleen, hæmaturia if the kidney is affected, or sudden coma followed by paralysis if the brain is involved. The emboli contain organisms, and therefore if the disease lasts long enough abscesses may form at the site of infarction, but the duration is usually a short one, and death occurs after an illness of a few weeks.

In the subacute form blood cultures usually show the infection to be due to non-hæmolytic streptococci. The source of the infection is often obscure, but may be from the throat or from septic teeth. This variety of endocarditis is often grafted upon previous heart disease, either rheumatic or congenital, and is only met with in the later years of childhood. Of 76 cases collected by Rost and Fischer,[1] none was below five years of age.

The onset is a gradual one, marked by progressive weakness, loss of weight, and anæmia. The temperature is usually raised two or three degrees, sweating may be a pronounced symptom, and there may be shortness of breath. Emboli often occur in the fingers and toes, where they may give rise to small red painful nodes which subside in a few days ; petechial hæmorrhages appear in the skin and mucous membranes, there may be retinal hæmorrhages, and infarctions in the viscera may account for enlargement of the spleen, hæmaturia, hæmoptysis, or hemiplegia. The heart is enlarged, and if there has been previous heart disease the murmurs may change in character or fresh bruits may appear. The course is a downhill one, and in spite of temporary remissions death occurs after an illness of from several months up to a year or two.

The treatment in both forms is mainly symptomatic. In the subacute variety vaccines, intravenous chemicals (mercurochrome, mercury perchloride, etc.), and blood transfusions may be tried, but are usually disappointing.

Suppurative Pericarditis

Suppurative pericarditis is very much less common in childhood than rheumatic pericarditis, and is almost always secondary to other acute diseases such as pneumonia or osteomyelitis. The incidence is highest during the early years of childhood—of 100 cases analysed by Poynton, eighty-five occurred before the fourth year. At this age the majority are due to the pneumococcus, while later on staphylococcal infections are a more frequent cause. The amount of pus varies from a trifling exudate in the most rapidly fatal cases, to a large accumulation distending the pericardium.

Symptoms. The onset is difficult to detect because the condition arises as an extension of disease in a child already gravely ill. Vomiting usually occurs, and there is likely to be a rise in temperature and a disproportionate increase in the pulse rate. The complexion shows an increasing pallor or lividity ; dyspnœa is very apparent, and there may be attacks of cyanosis or syncope.

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[1] Rost, W. L., and Fischer A. E., *Amer. Jour. Dis. Child.*, 1928, **36**, 1144.

Of physical signs pericardial friction is of the greatest importance, although if the pus rapidly accumulates frictions may only be present for a few hours, and so are easily overlooked. Next in importance is an increasing area of cardiac dullness, extending upwards as well as laterally. There are two signs of particular value ; one is that the area of cardiac dullness may extend well beyond the apex beat, and the other is a flattening out or disappearance of the normal acute angle between the right border of the heart and the liver. Delicate examination is required to detect these signs, and they are especially difficult to elicit when pus forms in the pericardium as a complication of pneumonia or empyema.

As the collection of pus increases, the heart sounds tend to become more and more muffled, and the apex beat becomes softer and may even disappear, and there may be some bulging of the intercostal spaces over the heart. The shape of the cardiac shadow as seen on an X-ray film may be of considerable help in diagnosis, especially if there is an opportunity of comparing films taken on successive days.

Treatment. The correct treatment consists of free surgical drainage of the pericardium. Simple tapping may be undertaken in order to verify the diagnosis before operation, but is not sufficient for treatment. Paracentesis of the pericardium may be performed through the left costo-xiphoid angle or over the area of dullness outside the apex beat.

Chronic Adhesive Pericarditis (Pick's Disease)

This rare condition, first described by Pick in 1896, consists essentially of a progressive constricting fibrosis of the pericardium, accompanied sometimes by plaques of calcification, and often associated with a more extensive adhesive mediastinitis. Tuberculosis of the pericardium accounts for some of the cases, others begin as an acute pericarditis due to some unknown infection. Rheumatic pericarditis does not appear to be a forerunner, and the condition is clinically distinct from the much more common " adherent pericardium " which occurs as a part of rheumatic heart disease.

The symptoms are due to the embarrassment of the cardiac action, the heart being encased in an unyielding sheath. Chronic enlargement of the liver and persistent ascites are usual features, to which may be added severe shortness of breath, turgescence of the veins of the neck, and widespread œdema. The heart is not enlarged, and the absence of bruits helps to differentiate the condition from chronic rheumatic pericarditis. When calcified plaques form in the pericardium, X-ray examination of the chest will assist the diagnosis.

The course is spread over several years, but is a slowly downhill one. Encouraging results have, however, been recently reported by White [1] following upon resection of much of the thickened pericardium.

Idiopathic Hypertrophy of the Heart

Under this name Howland has described instances in young children in which dilatation and hypertrophy of the heart have occurred without obvious cause. The condition has been found in still-born infants, but more often life continues for months or a year or two, and at first there may be nothing in the clinical history to attract attention. Later on attacks of cyanosis and breathlessness occur. Examination shows only the signs of a considerably enlarged heart.

Post-mortem examination reveals diffuse hypertrophy of the heart, affecting all chambers, so that the organ may weigh two or three times the normal. The endocardium is unaffected. In some instances the enlargement is due to the myocardium being packed with glycogen, and the condition should then be regarded as part of Von Gierke's glycogen-storage disease (see p. 261) rather than as an idiopathic disorder. It is also necessary to exclude coarctation of the aorta as a possible cause.

DISEASES OF THE VESSELS

Essential Hypertension

This is almost always a disease of adult life, but isolated instances have been reported in childhood. The illness runs a course of a year or two, the symptoms consisting of severe headaches, bouts of vomiting, and cerebral attacks accompanied by coma or fits, and followed by temporary blindness or paralysis. Examination of the retinæ may show flame-shaped hæmorrhages, patches of pale exudation, and swelling of the discs. Evidence of kidney involvement is indicated by albuminuria, and perhaps hæmaturia, and a lowered power of concentrating urea. The blood pressure is invariably raised, and may be over 200 mm. Hg.

The main pathological feature consists of hypertrophy of the media of the small arteries, the vessels of the kidneys being affected simply as part of a more widespread change. The aorta and large arteries may show atheromatous patches.

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[1] White, P., *Lancet*, 1935, ii., 539.

Isolated instances have also been recorded (Lightwood 1932) in which vascular changes similar to those just described have been associated with widespread calcification of the diseased vessels, making them hard and shotty to the feel, and easily identified in an X-ray film.

Aneurysm

Aneurysm of the aorta and large arteries is very exceptional in children, the few cases on record being usually associated with malformation of the large arteries, especially aortic coarctation. Septic and embolic arteritis and atheroma are rare causes. Instances have been recorded (Evans, Sheldon, J. H.) in which the aorta has shown considerable expansion during life owing to a high blood pressure, so much so as to give physical signs of an aneurysm, and yet after death it has been found to have returned to a normal calibre.

Raynaud's Disease

This also is a rare condition in childhood, although instances have been recorded in infants of only a few months old. The clinical picture consists of recurrent attacks of vascular spasm affecting the fingers or toes, which at first become cyanosed, swollen, and painful, and later are cold and insensitive. Recovery usually occurs after a few hours or days, but in more severe instances gangrene of the extremities may result. The condition is generally symmetrical.

The cause is uncertain. Exposure to cold is a factor, and chronic toxic absorption from the bowel may also play a part. Occasionally the attacks are associated with hæmoglobinuria, which should always prompt an examination for congenital syphilis.

Treatment consists of warmth to the extremities, attention to the general health, and anti-syphilitic treatment when there is evidence of that infection.

CHAPTER XVIII

DISEASES OF THE BLOOD, SPLEEN, AND LYMPHATIC SYSTEM

DISEASES OF THE BLOOD

Introduction

CONSIDERABLE alterations take place in the cytology of the blood during the first few weeks after birth, indeed the normal adult characters are not entirely assumed for some years. At birth the red cells number from 6 to 7 million per c.mm., and the hæmoglobin stands as high as 140 per cent. Of the red cells, a high proportion are nucleated—a feature which is normal to fœtal blood. During the first ten days a rapid destruction of red cells takes place, so that by the end of this period the red cell count has fallen to 4½ to 5 million per c.mm., the destruction being largely at the expense of the immature nucleated forms. One effect of the hæmolysis is to cause a temporary rise in the bilirubin level of the serum, which accounts for the brief physiological jaundice so common during the first week after birth. An excessive destruction of red cells is likely to occur in infants born prematurely, and in those of low birth-weight, and may lead in a few weeks to a recognisable degree of anæmia.

The hæmoglobin also falls rapidly after birth, reaching its lowest level at the end of the second month. At this age it averages in breast-fed infants about 73 per cent., and remains at this level until the second year, when it gradually rises to about 86 per cent.[1] The hæmoglobin level tends to be a little lower in artificially reared infants than in those fed from the breast.

During the first five or six years the white cell count is higher than the normal adult level, and except for the first few days after birth the excess of white cells is almost entirely due to lymphocytes. Within a day or two of birth the white cells number up to 25,000 per c.mm. By the end of the first year they have fallen to 15,000 per c.mm., of which 60 to 65 per cent. are lymphocytes. By the fifth or sixth year the count has fallen to about 10,000 per c.mm., of which about 40 per cent. are lymphocytes.

The reticulocyte count, the number of blood platelets, the fragility of the red cells, the coagulation time, and the bleeding time are the same in children as in adults.

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[1] Mackay, H. M. M., *Arch. Dis. Child.*, 1933, 8, 221.

ANÆMIA

Anæmia implies either a reduction in the number of red cells, or a diminution in the amount of hæmoglobin, or a mixture of both. In order that the red cells shall be maintained in normal numbers and that hæmoglobin shall continue to be formed, the bone marrow must be actively engaged in forming large nucleated primitive red cells (megaloblasts), and converting them through the stages of normoblast and reticulocyte to the finished article—the erythrocyte or red cell; for this to take place a factor must be present which is formed by the combination of an intrinsic element present in normal gastric juice with an extrinsic element which is closely allied to vitamin B_2, and in addition there must be an adequate supply of iron, copper, vitamin C, and thyroxin.

Broadly speaking, anæmia may be brought about in two ways, either from deficiency of one or more of the elements necessary for blood formation—deficiency anæmia; or from excessive destruction of red cells—hæmolytic anæmia. The anæmias of childhood classified in this way are disposed in the two groups as follows :—

Deficiency Anæmias.

Nutritional anæmia.
Anæmia of prematurity.
Anæmia of scurvy.
Anæmia of cœliac disease.
Osteosclerotic anæmia.
Anæmia of leukæmia.
Pernicious anæmia.
Aplastic anæmia.

Hæmolytic Anæmias.

Congenital hæmolytic anæmia (see p. 28).
Acute hæmolytic anæmia (Lederer).
Von Jaksch's anæmia.
Acholuric jaundice.
Cooley's anæmia.
Sickle-cell anæmia.

In a classification of this sort there is bound to be a certain amount of overlapping; for instance, the anæmia of prematurity is in part hæmolytic as well as in part a deficiency anæmia, and there is a large group of anæmic conditions, usually designated as secondary anæmia, such as the anæmia accompanying acute rheumatism, diphtheria, and septic or toxic states, which may belong to both groups.

DEFICIENCY ANÆMIAS

Nutritional Anæmia

This type of anæmia is characterised by a great reduction in hæmoglobin, which may fall as low as 30 or even 20 per cent., with a low colour index, and is due to an insufficient supply of iron, and probably copper as well. In a previous chapter the low iron content of human and cows' milk has been pointed out, and until other iron-containing foods come to form a regular part of the diet the infant has to rely on the stores of iron accumulated in the liver during fœtal life. If the storage is inadequate, through such causes as an iron deficiency in the mother, twin pregnancy, or premature birth, the infant is likely to develop anæmia within a few months of birth, and the greater the shortage of iron the earlier will the anæmia appear. Or a shortage may come about because a purely milk or cereal diet is continued too long, and iron-containing foods like meat broths and green vegetables are not given ; anæmia is then likely to become apparent before the end of the first year.

Symptoms. The outstanding symptom is pallor, which varies from a slight paleness of the mucous membranes to the most severe blanching. The spleen can sometimes be felt one or two fingersbreadth below the costal margin, but neither the liver nor the lymphatic glands are enlarged. The physical development is not appreciably impaired and the weight is generally normal. One result of the anæmia is to render the infant more liable to catarrhal infections such as bronchitis and otitis media. The anæmia of rickets is also nutritional, and is due to the fact that a diet which is likely to cause rickets is also likely to be deficient in iron.

In severe instances the red cells may be considerably reduced in number, although never to the same degree as the hæmoglobin. They are also smaller than normal, and the anæmia is therefore of the " hypochromic microcytic " type.

Treatment. The essence of treatment is to raise the hæmoglobin level by supplying iron. The response to treatment is very satisfactory, and even when the anæmia appears profound a steady recovery may be confidently expected. Of the various preparations of iron, iron and ammonium citrate is one of the best, grs. 2 to 5 being given thrice daily in a simple mixture [1] such as :

℞ Ferri et ammonii citratis gr. v.
Glycerini m. x.
Aquam ad ʒi.

Ferrous sulphate (gr. 3 ter die) or reduced iron (gr. 1 ter die) is also satisfactory. Of course the iron will darken the stools, and the mother should be warned of this.

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[1] The iron preparation can conveniently be added to the milk feeds. Hæmolac (Cow and Gate) is a dried milk with added iron.

The important part which copper plays in enabling inorganic iron to be built up into hæmoglobin has recently been realised, and it is of interest that a storage of copper parallel to that of iron, although in much smaller amount, is accumulated in the fœtal liver.[1] Fortunately, most iron ores contain traces of copper, and therefore medicinal preparations of iron usually have traces of copper in them as well—this was so in 80 per cent. of preparations of iron.[2] It occasionally happens that treatment with iron seems to be without effect, and it is then probable that the preparation of iron is copper-free. To avoid this a minute dose of copper sulphate (gr. $\frac{1}{200}$ to $\frac{1}{150}$) may with advantage be added to the iron prescription.

While the treatment detailed above will cure nutritional anæmia, it should be supplemented by ensuring that iron-containing foods are added to the diet. An ounce or so of bone-and-vegetable broth (for preparation see p. 70) can be given daily to young infants, and the iron content of the broth will be enhanced if prepared with bones that have small pieces of meat attached to them. A valuable broth may also be prepared by simmering and straining finely minced fresh liver, the virtue of the broth depending on its iron and not on any specific property of the liver. After six months of age a dessertspoonful of puréed spinach or sieved green vegetable may be given each day, together with raw meat juice.

Transfusions of blood are seldom necessary, but if the anæmia is profound a small transfusion may be given at the outset of treatment.

Anæmia of Prematurity

The destruction of red cells and reduction of hæmoglobin which normally takes place at birth continues in the prematurely born infant for some weeks, until after two or three months the red cells may be as low as 3,000,000 and the hæmoglobin be down to 50 per cent. It is difficult to say why this should be, but at all events the mere fact of prematurity implies a fœtal storage of iron below the normal, and, when once the anæmia has been produced, although the red cell count slowly rises the hæmoglobin lags behind and the clinical picture becomes similar to that of nutritional anæmia.

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[1] Ramage, H., Sheldon, J. H., Sheldon, W., *Proceedings of the Royal Society*, B, vol. 113, 1933, p. 308.

[2] Sheldon, J. H., and Ramage, H., *Quart. Jour. Med.*, 1932, New Series, 1, 1, 135.

The treatment is the same as for nutritional anæmia. In a case of any severity a small blood transfusion may be given.

Anæmia of Scurvy

Anæmia is often an early symptom of scurvy, although its significance may not be appreciated until other more positive evidence of the disease has appeared. The blood picture is similar to that of nutritional anæmia, but therapy with iron alone is ineffective, although when vitamin C is given in combination with iron the blood picture rapidly improves.

Anæmia of Cœliac Disease

Anæmia is a very constant symptom of cœliac disease, and is nearly always of the hypochromic microcytic type, being similar to other nutritional anæmias, and responding to iron therapy. Instances have, however, been recorded in which the colour index has been up to unity and the mean diameter of the red cells has been increased—hyperchromic megalocytic anæmia. Parsons found 3 examples of this type of anæmia in 19 instances of cœliac disease. The treatment of this particular form of anæmia lies in giving vitamin B, a suitable preparation being a teaspoonful of marmite three times a day, together with desiccated gastric mucosa. In practice, if iron therapy proves ineffective in overcoming the anæmia of cœliac disease, treatment with vitamin B should then be employed.

Osteosclerotic Anæmia and Leukæmic Anæmia

Any generalised disease which invades the bone marrow may be expected to produce anæmia by crowding out the blood-forming elements. In the rare condition of osteosclerosis fragilis generalisata (Albers-Schonberg disease) described on p. 608, the bone marrow gradually becomes encroached upon and converted into solid bone, with resulting severe anæmia. The grave anæmia that occurs in leukæmia is explicable in the same way by the marrow being packed with the leukæmic cells.

Pernicious Anæmia

Although pernicious anæmia is a fairly common form of deficiency anæmia in adults, most authorities agree that it does not occur in childhood, the evidence of the reported cases not being sufficiently convincing. The criteria required to support a diagnosis of pernicious anæmia should be :—

(1) Hyperchromic megalocytic anæmia, that is to say, an anæmia in which the colour index is up to or above unity, and the average diameter of the red cells is increased. (2) Achlorhydria. (3) A response to treatment by liver or gastric mucosa as shown by a rapid rise in the reticulocyte count. There can be no doubt that pernicious anæmia is very rare in childhood, but isolated instances have been recorded, the youngest being Faber's [1] case in an infant aged nine months.

Aplastic Anæmia

This condition is characterised by progressive anæmia without any evidence of repair on the part of the bone marrow—there is no reticulocytosis, and except perhaps in the early stages there are no nucleated red cells in the circulation. In many cases the cause is unknown ; in others there is a preceding acute infection such as tonsillitis, or there may be a focus of chronic infection in the throat or elsewhere, in which case the blood-forming centres have presumably been destroyed by infective or toxic influences. A similar condition is known to follow poisoning by benzol or trinitrotoluene, and to occur after over-exposure to X-rays. At post-mortem examination the marrow of the long bones is bloodless and yellow, and there are no accumulations of hæmosiderin in the tissues.

Symptoms. The onset is as a rule insidious, and the child is likely to be brought on account of increasing paleness, listlessness, giddiness, or shortness of breath. That a severe degree of anæmia is present is at once apparent, the child looks waxy and blanched, and there may already be petechial hæmorrhages in the skin, inside the mouth, and on the retina. The liver and spleen may be slightly enlarged, the latter just projecting beyond the costal margin. The temperature is often raised two or three degrees. As in any severe anæmia, the heart may be dilated, its rate is increased, and a systolic murmur may be audible at the apex and base and over the vessels in the neck.

Examination of the blood reveals a profound degree of anæmia. The red cells may number less than 1,000,000 per c.mm. and the hæmoglobin may be below 20 per cent. As a rule the white cells are also low, counts of from 1,000 to 5,000 per c.mm. being usual. The absence of normoblasts and reticulocytes confirms the diagnosis.

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[1] Faber, H. K., *Amer. Jour. Dis. Child.* 1928, **36,** 1121.

Diagnosis. This is made on the characters of the blood picture, and on the lack of response to treatment. The differential diagnosis from the acute hæmolytic anæmia of Lederer turns largely on the effect of blood transfusion, which in the latter condition is curative and evokes a reticulocytosis and normoblastic response, but is merely of temporary benefit in aplastic anæmia. There may also be difficulty in distinguishing aplastic anæmia from leukæmia, especially the variety called "aleukæmic leukæmia," but in the latter the spleen and superficial lymphatic glands are enlarged, normoblasts are usually present, and the differential white count shows a relative excess of leukæmic cells.

Treatment. The outlook is thoroughly bad, and the course is seldom longer than one or two months. The administration of iron, bone marrow preparations, and liver is without effect. Blood transfusion seldom give any but the most fleeting benefit, but if there is any evidence that the bone marrow has been stimulated by them, as indicated by the appearance of reticulocytes or nucleated red cells in the circulation, small repeated transfusions should be given. The effect of transfusion of blood should be carefully watched before hope is abandoned.

HÆMOLYTIC ANÆMIAS

Acute Hæmolytic Anæmia (Lederer)

This is a type of anæmia which was first recognised as an entity by Lederer [1] in 1925. In affects both children and adults, and develops very rapidly, so that within two or three weeks the red count may be as low as 1,000,000 per c.mm. and the hæmoglobin is reduced to an almost equal extent, the colour index being up to or slightly above unity—in other words, the anæmia is hyperchromic. The presence in the circulation of reticulocytes and nucleated red cells indicates that the marrow is active, while the hæmolytic nature of the anæmia is shown by an icteric tinge of the skin, urobilin in the urine, and sometimes bilirubinuria. Leucocytosis may occur, figures as high as 45,000 per c.mm. having been recorded. The spleen is usually palpable, petechial hæmorrhages and bruising may appear in the skin, and the temperature is likely to be raised a few degrees.

Treatment is an urgent matter. The beneficial effect of blood transfusion is usually striking, for after a single transfusion the marrow responds vigorously, as is shown by the sharp rise in the reticulocyte count. Small transfusions should then be repeated at intervals of a few days, until the blood picture approaches normal.

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[1] Lederer, M., *Amer. Jour. Med. Sc.*, 1925, **170,** 500.

The cause is not definitely known, but symptoms of acute intestinal disturbance such as vomiting or diarrhœa may occur at the onset, suggesting that the bone marrow may be injured by some toxic-infective process. Parsons[1] has suggested that the acute hæmolytic anæmia of Lederer and aplastic anæmia are essentially one and the same disease, response to treatment in the one case and not in the other being a reflection of the degree of injury sustained by the bone marrow.

Von Jaksch's Anæmia (Splenic Anæmia of Infants ; Anæmia Pseudo-leukæmia Infantum)

Much controversy has centred round this condition, some holding it to be a separate disease-entity, others maintaining that it is simply the result of any prolonged injury to the blood-forming organs. It has no relation to leukæmia, nor to the splenic anæmia of adults (Banti's disease), but should probably be regarded as a subacute form of hæmolytic anæmia brought about by various toxic or infective processes.

Symptoms. The age incidence lies between six months and three years The condition has been several times recorded in both of twins. The onset is insidious, and by the time the child is brought for examination the complexion is already very pale, and the liver and spleen are considerably enlarged, especially the latter, which may occupy almost the whole of the left half of the abdomen. Both organs are smooth, firm, and free from tenderness. The temperature is likely to be raised two or three degrees, but apart from the anæmia the amount of general disturbance is slight, and the child is usually plump. Signs of rickets are often present, and occasionally the Wassermann reaction is positive, but there is no good evidence that either rickets or syphillis are directly causative. Œdema, purpuric hæmorrhages into the skin and mucous membranes, and loud hæmic bruits may all be present in the more severe cases.

The blood picture shows the red cells to be reduced to 1 to 2 million per c.mm. and the hæmoglobin is at a correspondingly low level, the colour index being usually below unity. Nucleated red cells, poikilocytosis, and polychromasia are commonly seen in the blood film. The white cell count is raised, and is often between 30,000 and 40,000 per c.mm., with a relative lymphocytosis. A typical feature is the presence of myelocytes up to 6 or 8 per cent.—never high enough to cause confusion with leukæmia.

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[1] Parsons, L. G., and Hawksley, J. C., *Arch. Dis. Child.*, 1933, 8, 184.

Prognosis. This is much better than the appearance of the child would lead one to suppose. Roughly two-thirds of the cases make a gradual and complete recovery, although the spleen may remain palpable for several years. Death is usually the result of intercurrent broncho-pneumonia or diarrhœa. In fatal cases the fibrous tissue of the spleen is increased and the capsule is much thickened, the bone marrow is hyperactive, and extra-medullary centres of hæmopoiesis may be present in the liver, kidney, and elsewhere.

Treatment. There is no specific treatment. Such general measures as rest, fresh air, sunlight, and a nourishing diet are important, and if there is evidence of rickets or syphilis the appropriate treatment should be actively pursued. Iron—as iron and ammonium citrate (gr. 5 ter die) or reduced iron (gr. 2 ter die)—should be persisted with, and may be combined with one or two minims of Fowler's solution (liquor arsenicalis). Blood transfusions are of benefit in severe cases, but splenectomy is too great a risk to be justifiable, nor is it indicated, seeing that recovery usually occurs without it.

Acholuric Jaundice

Acholuric jaundice is frequently both hereditary and familial, affecting successive children of a family, although isolated instances also occur. Boys and girls are equally liable to be affected.

The symptoms are brought about by excessive destruction of red cells, although it is uncertain why hæmolysis takes place. The fragility of the red cells is increased, as may be shown by submitting them to various strengths of hypotonic saline solution. Red cells do not normally begin to hæmolyse until exposed to a saline solution of a strength of 0·42 per cent., and hæmolysis is complete in a strength of 0·35 per cent. In acholuric jaundice hæmolysis begins in saline solutions of a strength much nearer that of normal saline—in severe cases hæmolysis may be induced in saline at a strength of 0·6 per cent. Cockayne has pointed out that the fragility of the red cells, as tested in this way, cannot always be demonstrated, nor does it explain why the corpuscles should be fragile in the body, where they are not submitted to hypotonic saline. He suggests some inborn defect of the red cells rendering them unduly susceptible to the red-cell-destroying mechanism of the spleen.

Symptoms. Jaundice may be present at birth or appears shortly afterwards, and so may at first be confused with the physiological icterus of the newborn, although its persistence will soon rule out the latter. In other children the jaundice does not make its first appearance for several years. A usual feature is for the jaundice to deepen at intervals, and at such times the child may be sick, drowsy, and ill, while between times, except for a slight icteric tinge of the skin and conjunctivæ, the child may appear healthy and well-nourished. In others the jaundice may completely disappear in the intervals between attacks. The jaundice is hæmolytic in origin, and the stools remain bile coloured. The Van den Bergh test gives a positive indirect reaction. Whether the urine contains bile pigment or not depends on the depth of jaundice ; the urine is usually high coloured owing to urobilin, but only contains bile pigment during the more severe exacerbations.

Anæmia is usually present, and may sometimes be a much more prominent symptom than jaundice. Thus in a girl aged nine years the first symptom was a sudden anæmic crisis in which the red cell count fell to 1,040,000 per c.mm. Anæmia is generally more severe when the disease appears in infancy. The leucocytes are not affected.

The spleen is enlarged, extending two or three fingersbreadth below the costal margin, and is smooth and firm, and the liver may also be slightly enlarged.

Diagnosis. A history of recurrent attacks of jaundice should always suggest the condition, and should lead to an examination of the fragility of the red cells in order to confirm the diagnosis. A history of jaundice in other members of the family, the presence of bile in the stools throughout the attacks, and the enlarged spleen are other features that aid the diagnosis.

Prognosis. The outlook as regards life is favourable except in infancy, when the severity of the anæmia may make the prognosis grave. Otherwise the condition is compatible with long life. As in adults, so in children cholelithiasis may be a complication.

Treatment. Removal of the spleen offers the only prospect of cure, and the operation should be recommended when the attacks of jaundice are either frequent or severe, or when the anæmia is of a degree sufficient to cause invalidism. A pre-operative transfusion of blood is advisable. Following the operation, the fragility of the red cells often becomes normal, although this is by no means invariable, but nevertheless the attacks of jaundice cease, the anæmia gradually recovers, and the child becomes more lively and active. It is only in the slighter forms, when attacks of jaundice are fleeting or occur only at long intervals and the general health seems unimpaired, that splenectomy is not called for. Apart from removal of the spleen, there is little that can be done. Drugs are without effect.

Cooley's Anæmia [1]

This is a form of familial hæmolytic anæmia apparently confined to children derived from the stock of the Mediterranean littoral. The anæmia begins in infancy, and the blood picture is characterised by a considerable number of erythroblasts and a high reticulocyte count, indicating that the marrow is hyperactive ; in fact, the marrow may be so hyperplastic as to cause enlargement of the malar and cranial bones. The spleen is considerably enlarged.

The disease runs a slowly progressive course, and is ultimately fatal. A typical instance was reported in this country by Moncrieff and Whitby, and closely analogous cases have been described.

Sickle-cell Anæmia

This is a familial form of hæmolytic anæmia confined to the negro children of North America, and is characterised by a sickle or crescent shape of many of the red cells. The anæmia is variable, tending to wax and wane, and the spleen is usually palpable. No instance has been reported in this country.

SECONDARY ANÆMIA

Secondary or symptomatic anæmia, found in association with a great variety of acute and chronic diseases, is the most common form of anæmia in childhood. The blood picture shows a reduction chiefly of hæmoglobin, so that the colour index is below unity, although when the degree of anæmia is at all severe the number of red cells is lowered as well. The circumstances in which secondary anæmia is to be expected may be classified as follows :—

1. *Acute infections,* such as diphtheria, pyelitis, empyema, and acute rheumatism—especially when pericarditis develops.

2. *Chronic infections,* such as syphilis, tuberculosis—particularly when of the abdominal variety, tonsillar infection, otorrhœa,

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[1] Cooley, T. B., *Amer. Jour. Dis. Child.*, 1933, **33**, 786.

dental abscesses, chronic osteomyelitis, and chronic infection of the urinary tract.

3. *Chronic intoxications*, such as lead poisoning, chronic indigestion, constipation, and with intestinal parasites.

4. *Secondary to hæmorrhage*, and to such hæmorrhagic diseases as purpura and hæmophilia.

5. *Malignant New-growths.*

Secondary anæmia may be caused in various ways, for instance in acute rheumatism and diphtheria the anæmia may develop so rapidly as to point to a hæmolytic process at work, while in chronic alimentary disorders one or more of the various factors concerned in the formation of blood may be deficient in the diet or defectively absorbed. Direct loss of blood in the hæmorrhagic diseases is a further factor.

Symptoms. Secondary anæmia is itself a symptom, yet may cause additional symptoms which disappear when the anæmia is remedied. Among such may be mentioned breathlessness, too rapid fatigue, headache, fainting attacks, giddiness, and blurring of vision. The pulse rate is likely to be increased, and a systolic murmur may be audible over the apex and base of the heart and over the vessels of the neck.

Treatment. The first concern must be to treat the primary condition. For the anæmia a nourishing diet which includes meat, meat broths, liver soup and fresh vegetables should be given. Of drugs, iron, to which a trace of copper may be added, should be prescribed, and arsenic may be usefully incorporated. Some such prescription as the following will serve :—

℞ Ferri et ammonii citratis gr. v.
Liquoris arsenicalis m. i$\frac{1}{4}$.
Cupri sulphatis gr. $\frac{1}{200}$.
Glycerini m. x.
Aquam ad ℥i.

Blood transfusion is only required where the red cell count has fallen below 2,000,000 per c.mm. A change of air to the seaside is usually invaluable in accelerating recovery.

Polycythæmia

The most common cause of an absolute increase in the number of red cells in childhood is congenital heart disease, particularly when it is associated with cyanosis and finger-clubbing, as in congenital pulmonary stenosis. It may also be met with in chronic pulmonary disease. A rise in the red cell count above the normal figure may also occur from concentration of the blood, and thus may be met with in severe infantile diarrhœa, but this is not a true polycythæmia.

Primary Polycythæmia (Erythræmia; Osler-Vaquez Disease). This is a disease which usually produces its symptoms in middle age, but isolated instances have been reported in the later years of childhood. The condition is due to overactivity of the bone marrow. The red cell count may rise to between 8 and 12 million per c.mm. and the hæmoglobin may be raised to a corresponding extent. In children the symptoms have consisted of an unusually rubicund complexion, giddiness, breathlessness, and clubbing of the fingers. The spleen does not usually become palpable until after childhood.

Treatment. X-ray exposures applied over the long bones may lead to improvement. Splenectomy is not indicated.

Leukæmia

Although leukæmia may occur at any age, in childhood the disease shows certain peculiarities of which the most striking is its rapid course. Chronic myeloid and chronic lymphatic leukæmia are rarities in children.

The outstanding feature of leukæmia is the appearance of immature forms of white cells in the circulation, and in general terms the more immature the cells the more acute is the course of the disease. This is exemplified in the acute leukæmia of childhood, for often the prevailing white cell in the blood is a large cell with a large nucleus and a clear cytoplasm, so primitive that it is extremely difficult to decide whether it should be regarded as a lymphoblast or a myeloblast, nor do special stains such as the oxydase stain solve the difficulty, and therefore to attempt to divide such cases on hæmatological grounds into acute lymphatic and acute myeloid types is often impossible. A differentiation on clinical grounds is also deceptive, for most cases show both a general enlargement of lymphatic glands and considerable enlargement of the spleen, partaking of features which characterise both chronic lymphatic and chronic splenomedullary leukæmia. For these reasons it is preferable to speak of one disease, "acute leukæmia," and to picture chronic lymphatic and chronic myeloid leukæmia as the two extremes towards which acute leukæmia may move when it appears in a more chronic form, and when the leukæmic cells appearing into the circulation are less immature.

The cause of leukæmia is at present unknown.

Etiology. Leukæmia has been recorded as early as three weeks of age. In two infants observed by the author, both aged eight months, the course was very rapid, the first symptom—anæmia—having only been noticed three weeks before death. Male children are a little more often affected than females.

Symptoms. The onset is generally insidious, but occasionally dates from some acute infection such as tonsillitis or measles. Pallor and listlessness are first noticed, and there may be a complaint of abdominal pain, or of aching limbs caused by leukæmic infiltration under the periosteum of the long bones. Vomiting is a frequent symptom. As the disease progresses the skin becomes a pale waxy colour or may appear lemon-tinted, breathlessness increases, petechial hæmorrhages and bruising appear in the skin, and hæmorrhages may occur from the mucous membranes, accounting for severe epistaxis, hæmatemesis, or melæna. Blood may be passed in the urine, and small retinal hæmorrhages can often be found. Swelling of the gums and ulceration of the mouth and lips often develop towards the end of the disease, rendering the breath unpleasant. Leukæmic infiltrations occasionally form in the skin, giving rise to little nodules about the size of a pea. The temperature is raised, and may swing between 100° and 103° F.

The spleen is almost invariably enlarged, and may extend below the umbilicus, but it may not become palpable until quite late, and rarely is not to be felt at all. The liver also enlarges, but to a less extent than the spleen. The superficial lymphatic glands in the neck, axillæ, and groins are enlarged usually to the size of marbles, and are discrete and firm ; occasionally they are much larger, causing a collar-like appearance round the neck, or, when situated in the mediastinum, giving rise to symptoms of pressure on the trachea and large veins. The pulse rate slowly rises, until near the end it may be almost uncountable, the heart dilates, and loud murmurs become audible over the præcordia and vessels of the neck.

The Blood. Although the diagnosis of leukæmia turns on the character of the white cell count, the progressive anæmia is for the patient an equally important feature, indeed the ultimate cause of death lies in the grave anæmia rather than in the changes in the white cells. The red cells rapidly diminish in number, and are likely to be in the neighbourhood of two million by the time the child first comes under observation, while before death they may drop below a million. The hæmoglobin is even more reduced, and the colour index is usually well below unity.

The white cells vary considerably in total number, in fact from the standpoint of diagnosis the differential white count is of much more importance than the total white count. Usually they number between 15,000 and 40,000 per c.mm., but may rise as high as 200,000, while in the aleukæmic variety they may fall to 2,000 or less. The differential count shows invariably a high percentage of immature white cells. In some cases myelocytes with a granular cytoplasm are present in numbers from 30 to 70 per cent., in others small lymphocytes predominate up to 90 to 97 per cent., the blood picture falling clearly into myeloid and lymphatic types. In others the predominating cell has resemblances to a large lymphocyte, and may be either a lymphoblast or a myeloblast. This is particularly likely to be the case when the total white cell count is low.

Aleukæmic Leukæmia (Leukanæmia). This variety of acute leukæmia is characterised by a remarkably low total white cell count, but otherwise the clinical picture as regards anæmia and enlargement of glands and spleen is identical with the ordinary form of leukæmia. An instance was that of a boy aged two years whose blood picture eight days before death showed a white cell count of 2,200 per c.mm.; 95 per cent. of these cells were lymphocytes. Although the aleukæmic condition of the blood may persist until death, it may be but a phase in the course of leukæmia, and successive blood counts may show the white cells mounting again. Thus in a boy of five years a blood count one month before death showed only 2,100 leucocytes per c.mm., and of these 58 per cent. were lymphocytes. A week before death the white cells had risen to 16,400 per c.mm., and the differential count showed the lymphocytes to be 97 per cent.

Diagnosis. The presence of a general enlargement of lymph glands and spleen in a severely anæmic child will prompt an examination of the blood, and the differential white cell count will then generally settle the matter. The blood disease most likely to be mistaken for leukæmia is Von Jaksch's anæmia, but in the latter the chronicity of the anæmia, the absence of enlarged glands, and the presence of a small percentage of myelocytes are important distinguishing features. The differentiation is of prognostic importance, because the outlook in Von Jaksch's anæmia is usually favourable. The hæmorrhages into the skin may at first sight suggest toxic or infective purpura, especially as in purpura the spleen may be palpable, but there is no general enlargement of lymph glands, nor does the blood picture resemble that of leukæmia. When leukæmia begins with limb pains, the anæmia and cardiac bruits may raise the question of acute rheumatism. The limb pains of leukæmia are, however, over the long bones rather than in the joints, while a complete examination will reveal other features of leukæmia such as the enlarged spleen and lymph glands. X-ray examination of the long bones may show the periosteum raised by leukæmic infiltration.

Pathology. Post-mortem examination shows a general bloodlessness, with petechial hæmorrhages under the serous membranes and beneath the capsules of most organs. The spleen is soft and of a deep purple colour, the lymph glands are pinkish-grey, and in both there is a heavy infiltration of leukæmic cells. The bone marrow of the long bones is of a reddish-grey colour, and contains masses of leukæmic cells and also an increased number of nucleated red cells. Numerous small foci of leukæmic cells are also present in most organs, notably in the liver, kidneys, lungs and heart.

Prognosis. The disease is invariably fatal, and in childhood the course is generally about two months, but in the most acute cases may be as short as three weeks. The remissions which may be hoped for in the chronic leukæmias of adults do not occur in the acute form in childhood.

Treatment. There is no curative treatment. Blood transfusions produce only a fleeting benefit, and merely postpone the inevitable end by a few days. Splenectomy is useless, intramuscular injections of arsenicals have seemed to bring about a slight improvement, but it is only temporary. In cases that show a tendency to chronicity careful X-ray exposures over the long bones and spleen are sometimes beneficial, but in acute cases they are as likely to accelerate the disease.

Chloroma

This is a variety of acute leukæmia characterised by the formation of leukæmic tumours under the periosteum of the skull bones, especially around the orbits, and less often in the ribs, vertebræ, and viscera. At post-mortem examination the tumours often have a greenish hue, and histologically are composed of leukæmic cells.

The tumours that form in relation to the orbits are likely to cause severe headache, proptosis, papillœdema, and orbital hæmorrhages. The other changes of leukæmia, such as enlargement of the spleen and lymph glands, are present, and the blood picture is that of leukæmia.

Death takes place after a course of two or three months. There is no effective treatment.

Hæmophilia

This is an hereditary disease characterised by a tendency to prolonged bleeding from wounds of even a slight nature.

Etiology. The symptoms of hæmophilia are manifested only by males, while the transmission of the disease from one generation to the next takes place only through the female. In their exhaustive study of hæmophilia, Bulloch and Fildes [1] were unable to find satisfactory evidence of a female ever manifesting the disease.

Symptoms. The outstanding symptom is intractable hæmorrhage, and that the patient is liable to this has usually become clear by the end of the second year. The newborn child is exempt, but fatal oozing after the operation of circumcision has occurred at a few weeks of age. It is the relentless persistence of the bleeding, rather than any sudden and severe hæmorrhage, which is so characteristic.

Almost always the hæmorrhage is initiated by trauma. Thus picking at the nose may start an epistaxis which goes on for days, and superficial skin abrasions and dental extractions are common sources of bleeding. Surgical operations are, of course, highly dangerous.

Hœmophilic Hœmarthrosis. Hæmorrhage into the joints is one of the most serious complications. The elbows and knees are most often affected, because they are the joints most exposed to injury. The joint rapidly swells and becomes so very tender that the child refuses to move it. As a rule the skin over the joint does not show any discoloration. With complete rest the blood slowly becomes absorbed, but some degree of fibrous ankylosis follows, and osteophytic outgrowths may form at the edge of the articular surfaces, with permanent impairment of function. Once a joint has been affected, recurrences of hæmorrhage into it are likely to take place.

Examination of the blood may show anæmia if there has been a recent hæmorrhage. The principal feature is, however, an increase in the coagulation time, which may be prolonged from the normal two to four minutes up to as long as half an hour or more, and is due to a failure in the formation of thrombin. The bleeding time may be either normal or prolonged. The platelet count is normal.

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[1] Bullock and Fildes. "Treasury of Human Inheritance," 1911, Pts. V. and VI.

Diagnosis. This is not difficult if the child, a male, can be shown to be descended from hæmophilic stock. It not infrequently happens that children with recurrent purpura are mistakenly regarded as hæmophiliacs, and a history of a " tendency to bleed " may be raked up from their family history, but the correct mode of transmission of hæmophilia, namely, through the females with symptoms only in the male members, is not likely to be obtained. In purpura the bleeding time is prolonged, but the coagulation time is normal. The prolonged coagulation time of hæmophilia is a reliable diagnostic criterion.

Hæmophilic hæmorrhage into the knee may closely simulate a tuberculous joint, but the family history, the possibility of previous similar attacks, and the coagulation time of the blood are all points of distinction.

Prognosis. Attacks of bleeding vary in severity, but even from a slight abrasion the patient may gradually become exsanguinated. It is probable that less than half the cases live beyond puberty.

Treatment. Prevention of hæmorrhage must be ensured as far as possible by protecting the child from possible sources of injury, and by avoiding such common operations as phimosis, dental extraction, and tonsillectomy.

The local treatment of hæmorrhage is unsatisfactory. Styptics and adrenalin are only of passing benefit, but if the bleeding point can be seen a compress of fresh human blood may be applied. For the more severe and prolonged hæmorrhages blood transfusion should be employed. The treatment of hæmarthrosis consists of keeping the joint at rest on a splint, massage and passive movements being deferred for a fortnight or so lest fresh hæmorrhage is excited.

Recently Macfarlane and Burgess [1] have reported the valuable coagulant effect on hæmophilic blood of snake venom, and have applied it locally with most promising results.

Purpura

Purpura is a condition in which spontaneous hæmorrhages occur in the skin and mucous membranes. It should be regarded, at any rate in the majority of cases, as a symptom which may arise from various causes, and may be fairly compared with a symptom such as anæmia. It is met with throughout childhood, but, apart from the variety that is associated with cachectic states, is much more common after six years of age. The sexes are equally affected.

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[1] Macfarlane, R. G., and Burgess, B., *Lancet*, 1934, *ii.*, 985.

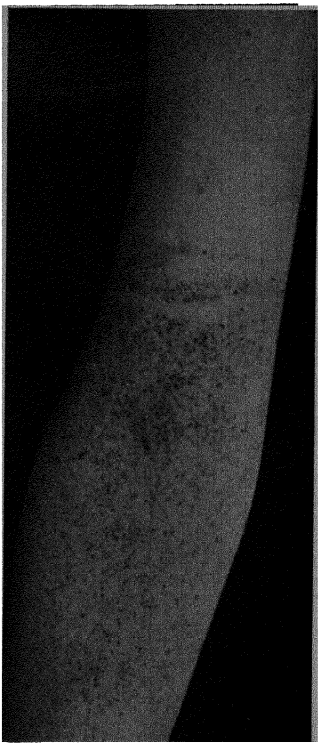

FIG. 64. Positive capillary resistance test on the right arm of a child suffering from purpura.

The mechanism at work in the production of the hæmorrhages is imperfectly understood, but it is more probable that the lesion is primarily in the vessel walls, which are injured sufficiently to allow blood to escape, than that there is a primary alteration in the physical properties of the blood. Thus the manner in which the hæmorrhages are dotted here and there is certainly more suggestive of multiple focal lesions of the vessels than of a general change in the composition of the blood, and the same interpretation applies to the patches of œdema which occur in some of the cases. Then, too, purpura is known to follow poisoning with endotheliolytic toxins such as are present in some snake venoms. The capillary resistance test points to the same conclusion. This test consists of applying a band round a limb sufficiently tightly to prevent the venous return ; in a healthy limb the capillaries can withstand the strain put upon them, and hæmorrhages do not appear, but in purpura after two or three minutes a crop of petechiæ may develop distal to the bandage. Recent investigations have suggested that the capillary resistance may be lowered by deficiency of vitamin P (citrin : hesperidin), a substance which is found in association with vitamin C.

In addition to a degree of anæmia which depends on the extent of the hæmorrhages, the salient feature of the blood picture in many cases consists of a reduction in the number of platelets, which may drop considerably below the normal figure of 200,000 –400,000 per c.mm., and in the severest cases may be almost entirely absent. The fall in platelets is, however, by no means an invariable accompaniment of purpura. The reduction in their number accounts for two other characteristics of the blood; the first is a prolongation of the bleeding time from the normal two to four minutes up to ten minutes or more (the coagulation time remains normal), and the second is a loss of the normal retractility of the blood clot. The significance of the low platelet count is uncertain, for it can hardly be held to initiate the hæmorrhages, even if it accounts for the prolonged bleeding time.

A classification of purpura according to the various factors that may be concerned in its production offers the simplest understanding of the condition.

1. *Toxi-infective Purpura.* This includes purpura associated with bacterial and other infections and bacterial toxæmia, and, in the writer's opinion, accounts for the majority of the cases.

In this group is the purpura which occasionally complicates the acute specific fevers, particularly diphtheria, small-pox, scarlet fever and measles, and which always adds to their seriousness. In other acute infections, notably meningococcal meningitis and typhus fever, a purpuric eruption forms a characteristic part of the clinical picture. Acute septic infections such as tonsillitis or dental abscesses are in childhood a frequent forerunner of purpura, preceding the eruption by an interval of a few days, and purpura may also be caused by bacterial emboli in septicæmic states and in infective endocarditis. It is not improbable that sources of chronic toxæmia such as an unhealthy state of the bowel and chronic constipation may also initiate an attack.

Purpura is a rare event in acute rheumatism, but may complicate severe and fulminating cases of rheumatic carditis. This must not be confused with the so-called "purpura rheumatica" (arthritic purpura; peliosis rheumatica; Schönlein's disease) which is probably an example of allergic purpura, and in which painful joint effusions and patches of angio-neurotic œdema are associated with a purpuric eruption. Whatever is the cause of these attacks, there is no satisfactory evidence to connect them with acute rheumatism, for they show no relationship with such definite rheumatic conditions as carditis and chorea, nor do the attacks respond to anti-rheumatic drugs. It is unfortunate that the name purpura rheumatica has been applied to them, for it is nothing but misleading.

2. *Toxic Purpura.* In addition to toxins manufactured in the body, exogenous sources of toxin such as drugs and snake

venom may give rise to purpura. The drugs that may do this include arsenic, iodides, phenazone and quinine.

3. *Cachectic Purpura.* The chronic wasting disorders of childhood are liable to be complicated by purpura. Thus in infancy it may be met with in the emaciated state following upon gastro-enteritis, in severe hypertrophic stenosis of the pylorus, and in the chronic stage of post-basic meningitis. In older children it may occur in cœliac disease, and in tuberculous enteritis and peritonitis. Cachectic purpura usually appears on the trunk or lower abdomen, especially in the folds at the base of the neck, round the axillæ, and in the groins. The hæmorrhages have a curious lilac tint.

4. *Purpura in Association with Grave Anœmia.* Petechial hæmorrhages in the skin, mucous membranes, and retina may accompany any of the grave anæmias of childhood. They are a usual feature of leukæmia and may also occur in aplastic anæmia, lymphadenoma, Von Jaksch's anæmia, and infantile scurvy.

5. *Allergic Purpura.* This is the type of purpura which may be associated with patches of giant urticaria (angio-neurotic œdema), painful effusion into joints (Schönlein's disease), and often with severe abdominal pain (Henoch's purpura). As a rule the platelet count is not diminished, the bleeding time is normal, and the retractility of the clot is unaffected. To this group also belongs the purpura which occasionally occurs in serum-sickness.

6. *Primary or Idiopathic Purpura.* That instances of purpura occur for which no predisposing cause can be found is quite certain, but whether they form a separate disease-entity, as has been suggested, is open to doubt. In addition to the absence of any traceable cause, these cases show a considerable reduction of platelets, a prolonged bleeding time, and a non-retractile clot. Moreover the purpura shows a decided tendency to recur, and may sometimes be almost continuous.

Clinical Features. In the common form of purpura the first symptom is the appearance of hæmorrhages in the skin, ranging in size from minute petechiæ to extensive bruises. Apart from the hæmorrhages the child may seem but little out-of-sorts, the temperature is scarcely raised, the appetite is unimpaired and there may be no trace of hæmorrhage from the mucosal surfaces. The smaller hæmorrhages fade and disappear in a week or so, but fresh points of bleeding may continue to appear for a few weeks. The larger bruises go through the usual colour changes of blue, green, and yellow, before finally disappearing. Although any part of the skin may be affected, bruises are most likely to appear over bony prominences such as the rim of the pelvis, in the neighbourhood of joints, on the shins, and over points of pressure such as the buttocks. It sometimes happens that the hæmorrhages quickly cease so long as the child is rested in bed, but fresh crops appear as soon as he is allowed to get up, as though the capillaries are unable to stand the extra strain imposed on them by gravity.

In more severe cases hæmorrhages occur from the mucous membranes as well as into the skin. Thus petechiæ may be found on the palate and inside the cheeks, the gums bleed easily, and nose-bleeding may be severe enough to require plugging. True hæmatemesis is rare, but blood which has been swallowed from the post-nasal space may be vomited and so simulate hæmatemesis.

Effusions of blood may take place into the wall of the intestine, and give rise to bouts of severe colicky pain, followed maybe by the passage of blood and mucus in the stools (Henoch's purpura). Confusion with acute intussusception is then very likely, and may lead to an unnecessary laparotomy. It may be added that the vigorous attempts of the intestine to rid itself of the hæmorrhagic effusion have been known to cause an intussusception. There are, however, two points which may help to prevent an error in diagnosis; one is that a careful scrutiny may reveal a few petechiæ in the skin, and the other is the association of this form of purpura with patches of angio-neurotic œdema or giant urticaria. These may be a few inches in diameter, and may occupy such odd situations as the forehead or over the chest. They arise quickly, are painless, pit on pressure, and, after persisting for two or three days, quickly absorb. They may precede the hæmorrhages by a day or so, or may coincide with them.

Mention has already been made of the effusions into joints which accompany some cases of purpura (arthritic purpura ; Schönlein's disease). Several joints may be affected, becoming swollen and tender, and the neighbouring skin may show an erythematous flush. The skin hæmorrhages may be so few as to form but a small part of the clinical picture. The symptoms of Schönlein's disease and Henoch's purpura sometimes occur in combination.

Bleeding into other organs is uncommon. A hæmorrhagic type of acute nephritis may occur, characterised principally by hæmaturia, and may sometimes persist for months after the original purpura has disappeared. Severe hæmorrhage into the eye leading to total loss of vision has been recorded, and fatal cerebral hæmorrhage has also occurred.

The spleen can occasionally be felt extending one or two fingersbreadth below the costal margin.

Course and Prognosis. The duration of an attack varies considerably, and a small number of hæmorrhages does not necessarily indicate a short course. Many cases clear up in a fortnight or so, but in others fresh hæmorrhages may continue to appear for months. A boy who came under the writer's care at the age of three years, and who was frequently seen for the next two years, was never entirely free of petechiæ and small bruises, but in spite of this his general health was not appreciably affected. More often the purpura tends to recur at intervals of a few weeks or months, and a child may have half a dozen or more attacks. Cases such as these are likely to fall into the " idiopathic " group, but there is often room for a lurking suspicion that the tonsils or some other focus of toxic absorption are at the bottom of the attacks.

Purpura Fulminans. This form of purpura is fortunately uncommon, for it is characterised by a rapid and fatal course. There may be a preceding history of a sore throat or of one of the specific fevers, but other instances arise for no apparent reason. The history is usually somewhat as follows:—the patient, generally under two years of age, begins his illness with a crop of petechiæ and bruises in the skin. There may be a sharp attack of diarrhœa, and within perhaps a few hours the child is pale, prostrate, and stuporous. The purpuric eruption rapidly increases, the stupor passes on to coma, and death follows after the illness has only lasted twenty-four to forty-eight hours. The occurrence of rapidly fatal hæmorrhage into the suprarenals in young infants (see p. 478), associated with a quickly spreading purpuric eruption, may also be looked upon as an example of purpura fulminans.

With the exception of purpura fulminans, the prognosis as regards recovery is almost invariably good.

Treatment.[1] Rest in bed is essential. This, with a mild purge, may be sufficient for mild cases. Septic foci should be dealt with either immediately or during convalescence according to the nature of the individual case. As to drugs, a combination of iron and arsenic is useful (for prescription see p. 446). Calcium salts, sometimes in combination with small doses of parathyroid gland (gr. $\frac{1}{10}$), have been recommended, but their benefit is seldom obvious. For more severe cases injections of horse serum or of polyvalent anti-streptococcal serum are sometimes beneficial, but better results are generally obtained by a transfusion of whole blood. Large doses of turpentine, such as one or two drachms, combined with at least the same amount of castor oil, were recommended by Eustace Smith, but the writer has had no experience of this treatment. The pain of Henoch's purpura is best relieved with opium ; to a child of five years pulv. ipecac. co. gr. 2, or tinct. opii m. 2, would be a suitable dose.

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[1] As judged by the improvement that may come about in the capillary resistance test, vitamin P is sometimes of benefit. It is sold under the name Hesperidin ; the dose should be 1 grm. daily.

In recent years removal of the spleen in carefully selected cases has given good results. The operation should never be undertaken when the disease is acute, but should be recommended in " idiopathic " cases which run a chronic or frequently recurring course, and in which the platelet count is diminished. Whether the spleen is palpable or not does not affect the decision to operate. Splenectomy is usually followed by a rapid rise in the platelet count, and although the number of platelets may subsequently fall again the tendency to hæmorrhage does not reappear. Provided that the cases are carefully chosen, the operation proves beneficial in about 80 per cent. (A. W. Spence). It should be preceded by a blood transfusion.

AFFECTIONS OF THE SPLEEN AND LYMPHATIC GLANDS

Hodgkin's Disease (Lymphadenoma)

Hodgkin's disease in childhood differs in no important respect from the disease as met with in adults. It affects boys more often than girls, and is rarely encountered before the second half of childhood. The cause is unknown. Some have held it to be of the nature of a new growth, others have regarded it as infective, although no organism or filterable virus has as yet proved its claim.

Symptoms. The onset is insidious, and the first symptom to attract attention is as a rule enlargement of the cervical lymphatic glands. These vary in size from a marble up to a pigeon's egg, and are at first firm, painless, and discrete, but in the later stages may become matted together. Other groups of glands gradually become affected, and big masses may become palpable in the axillæ and groins. Enlargement of the mediastinal glands may sometimes occur early, and by pressing on neighbouring structures give rise to cough, stridor, shortness of breath, dysphagia and dilatation of the superficial veins of the chest and neck, while involvement of the glands on the posterior abdominal wall may account for large irregular masses. The spleen as a rule becomes much increased in size, and may reach down to the umbilicus. It may remain smooth, or large masses of lymphadenomatous tissue may form in it, making the surface nodular and the edge irregular (hardbake spleen). The liver is likely to be similarly affected.

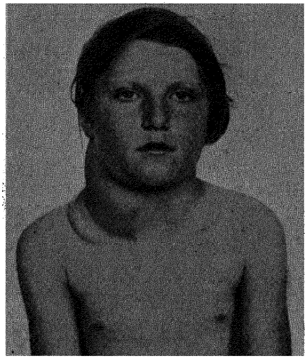

FIG. 65. Hodgkin's disease in a girl aged eleven years, showing a mass of enlarged glands in the neck.

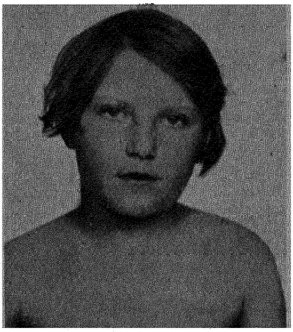

FIG. 66. The same patient as in Fig. 65, showing improvement after X-ray treatment.

As the disease progresses, anæmia becomes more and more obvious. The cervical glands may eventually form such a large collar-like mass as to give the patient a frog-like appearance. Effusions of clear serous fluid often form in the pleural and peritoneal cavities, and the legs may become œdematous owing to obstruction of the inferior vena cava. Rarely, nodules of lymphadenomatous tissue form in the skin, and compression-paraplegia may occur owing to involvement of the spine. The temperature is generally raised ; there may be a continuous fever of two or three degrees, or it may be remittent, but the most usual form is the Pel-Ebstein type in which the temperature is raised to 103° F. or so for periods of a week or ten days, separated by two or three weeks during which the temperature is normal.

The blood picture shows nothing characteristic. The red cells and hæmoglobin gradually fall as the anæmia becomes more marked, and as a rule there is a slight increase of leucocytes up to 15,000 to 20,000 per c.mm., but a leucopenia is present in roughly a quarter of the cases. The differential count is likely to show a slight increase of eosinophil cells.

Diagnosis. Localised enlargement of lymphatic glands, especially those in the neck, must be distinguished from tuberculous adenitis. Tuberculous glands tend to be matted together, and may break down into a soft fluctuating abscess; Hodgkin glands on the other hand are characteristically discrete, and do not become attached to surrounding structures. But these features are not always clear cut, and then if doubt exists a superficial gland should be excised for histological examination. The microscopical appearance of a Hodgkin gland shows a loss of the normal architecture, which is replaced by a homogeneous field containing a great increase of endothelial cells, some of which become multinucleated with three or four nuclei grouped in the centre of the cell; the fibrous trabeculæ of the gland are more apparent, and there may be a considerable increase of eosinophil cells.

Some cases of lymphosarcoma may resemble lymphadenoma, but the glandular enlargement is more rapid, while a general spread to other glands and enlargement of the spleen are points against sarcoma. Confusion with leukæmia will be prevented by the blood examination.

Prognosis. Lymphadenoma is invariably fatal. Occasionally fulminating cases occur which run a course of only two or three months, but generally the disease lasts for as many years. Remissions may occur under treatment with X-rays or arsenic, or there may be months in which the disease seems stationary, but sooner or later the condition advances again.

Treatment. When the disease is confined to the cervical glands, their surgical removal may give a temporary check, but usually within a few months other groups of glands become involved. Deep X-ray therapy and treatment with radium are sometimes followed by a reduction in size of the glandular masses and the spleen may shrink considerably, but the improvement is not permanent. Arsenic may induce remissions, and may be given as Fowler's solution, the dose being worked up from three or four minims to the limit of tolerance, or the arsenobenzol preparations may be given intravenously (see p. 660). Opiates should be used for the relief of pain.

Banti's Disease (Splenic Anæmia)

This is a chronic condition, characterised at first by considerable enlargement of the spleen and progressive anæmia with leucopenia, and followed after some years by cirrhosis of the liver, which gradually dominates the clinical picture and may cause considerable ascites. Hæmatemesis is sometimes the first symptom to attract attention. The hæmorrhage may be repeated, or may be severe enough to prove fatal. Wallgren [1] has described instances in childhood of great enlargement of the spleen associated with severe and fatal hæmatemesis, which might well have been regarded as examples of Banti's disease, but in which post-mortem examination revealed a thrombosis of the splenic vein as the cause of the illness.

Diagnosis. The disease is usually met with in adults, but isolated instances have been recorded in the late years of childhood. It is, however, so rare at that age that the diagnosis can only be made after a careful exclusion of all other possibilities. The condition bears no relation to the splenic anæmia of infants (Von Jaksch's anæmia), in which the age incidence, the increase of myelocytes in the blood, and the absence of hepatic involvement serve as distinguishing features. Gaucher's splenomegaly may be differentiated by its familial incidence and by finding the large "Gaucher" cells in the material from a splenic puncture. Congenital syphilis, massive tuberculosis of the liver and spleen, hepatic cirrhosis, and lymphadenoma are other possible sources of error.

Treatment. Removal of the spleen is the only satisfactory treatment, but to obtain the best results the operation must be carried out before the liver becomes involved.

DISEASES OF THE LYMPHATIC SYSTEM

Primary disease of the lymphatic system is rare, but occurs in the form of lymphosarcoma. The lymphatic glands may be affected as part of some general disease such as lymphadenoma or leukæmia, otherwise their morbid conditions arise almost entirely from local infective processes draining to the regional lymphatic glands.

Congenital dilatation of the lymph spaces occurs in a localised form (lymphangioma) and in a diffuse form (lymphangiectasis). The localised form is most likely to appear in the neck as a soft fluctuant tumour (cystic hygroma), but may also develop in other situations as, for instance, in the axilla or groin. Its treatment is surgical. The diffuse form may affect the tongue (macroglossia), or may cause gross enlargement of one or more limbs as in Fig. 67, sometimes giving to them an appearance of roll upon roll of fat. There are also cases in which the whole of one side of the child is involved, the arm and leg on the affected side being bigger in circumference and longer than the opposite limbs (hemi-hypertrophy). In some of these the skin has a patchy bluish appearance, indicating a mixture of nævoid and lymphangiectatic states.

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[1] Wallgren, A., *Acta Pediatrica*, 1927, **6** (Supplement).

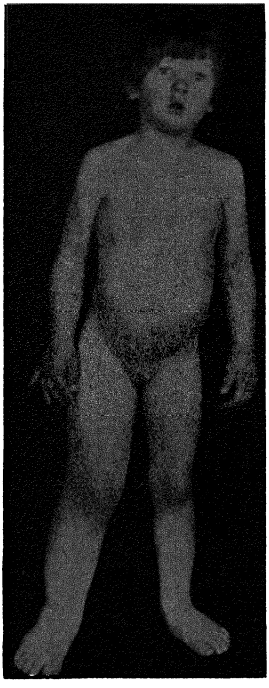

FIG. 67. Girl aged four years with lymphangiectatic enlargement of the right leg.

Tuberculous Cervical Adenitis

Tuberculous glands in the neck are most commonly met with in the early years of childhood. They are involved as the result of tuberculous infection of the tonsils, and the realisation of this has materially altered the treatment, for while some years ago surgical dissection of the glands was the usual procedure and the tonsils often escaped attention, at the present day the source of the infection is dealt with by removing the tonsils, and unless the glands are actually breaking down into abscesses they are left alone and allowed to heal. General dissemination of tuberculosis from the cervical glands is fortunately uncommon. The incidence of tuberculous glands in the neck is difficult to estimate, but it is probable that the condition is more often diagnosed than is actually warranted. There is certainly no doubt that it has become less common in recent years, and the explanation of this lies in the increasing practice of boiling or pasteurising cow's milk, and so rendering it free from tuberculous infection.

As a rule the enlargement of the glands takes place gradually, although any superimposed septic infection of the throat may cause them to swell rapidly. The "tonsillar" gland at the angle of the jaw is the first to be affected, followed by enlargement of glands lower in the neck. At first the glands are firm and scarcely tender, later they tend to become matted together, and eventually they may liquefy, and, becoming attached to the skin, form a red fluctuant swelling which if left alone will ultimately burst and leave a chronic sinus.

The diagnosis is simple enough when the glands have liquefied and formed a " cold " abscess, but before then it is often impossible to distinguish tuberculous from septic glands, nor does the naked-eye appearance of the tonsils help, because the tuberculous tonsil *in situ* has no distinguishing features. The final diagnosis may then have to be deferred until the tonsils have been removed, when a histological examination should be made ; if the tonsils are found to be tuberculous it is safe to assume that the glands are similarly infected. Importance is to be attached to the matting together of several glands, which is usual in tuberculosis, but may sometimes occur in subacute septic glands. When lymphadenoma is confined to the cervical glands, as it often is in its early stages, the discrete character of the glands is a valuable distinguishing feature. Lymphosarcoma is fortunately rare, but the rapid growth of the glands, the local infiltration, and the appearance of secondary growths will prevent error. The distinctive features of glandular fever are detailed elsewhere (p. 709).

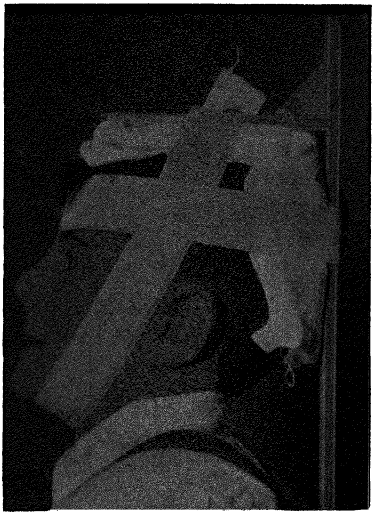

FIG. 68. To show the method of applying a splint for tuberculous glands in the neck.

Treatment. Treatment should begin with the removal of the tonsils. After the operation the child should be rested in bed until the temperature settles. It is often surprising how quickly a tuberculous gland will diminish in size after the source of the infection has been removed, but so long as the glands are large the neck should be immobilised ; this may be most conveniently carried out by means of splints (see illustration), and then if the temperature is not raised the child need not be confined to bed. Local operation becomes necessary in the event of the glands threatening to break down into an abscess.

General treatment is of great importance. A stay of several months at the seaside should be arranged, and during the winter months graduated exposures to ultra-violet rays should be given. The diet must be as nourishing as the child will tolerate, and in order to assist healing by calcification, 10 gr. Calcium Gluconate should be given in a simple mixture three times a day, together with a good cod-liver oil emulsion combined with malt and iron.

Generalised Tuberculosis of Lymphatic Glands

This rare condition may be met with in young children. The superficial glands in the neck, axillæ, and groins rapidly enlarge and become caseous, and there may be evidence of involvement of the mediastinal and deep abdominal glands. The outlook is grave, owing to the likelihood of a miliary or meningeal spread.

Lymphosarcoma

This is rare in childhood. The glands in the mediastinum or in the neck are likely to be the starting point. They rapidly enlarge, and are soft, slightly tender, and soon become attached to surrounding structures. When the disease occurs in the mediastinum symptoms soon arise from the local pressure effects, and include cyanosis, dyspnœa, dysphagia, and dilatation of the superficial veins on the chest wall.

Treatment by radium or deep X-rays may be tried, but as a rule the disease advances rapidly, and the child dies within a few weeks.

Mikulicz's Disease

This is a disorder characterised by enlargement of the salivary and lachrymal glands. It may be due to various conditions, of which leukæmia and lymphadenoma are in childhood the most usual and the most serious. There is also a benign form in which the glands remain swollen for several weeks and then slowly subside, but the swelling may recur. The swollen glands cause some disfigurement, and may be mistaken for mumps.

BLOOD TRANSFUSION

Blood transfusion has now become so firmly established as a therapeutic procedure that it is appropriate to close this chapter with a consideration of some of the points which arise when it is employed in children.

Indications. The most obvious indication for transfusion is after a severe loss of blood, as for instance after trauma or operation, or when severe internal hæmorrhage has taken place. It may be used in the hæmorrhagic disease of the newborn (hæmatemesis or melæna neonatorum), and has an advantage over blood given intramuscularly in that it not only checks the bleeding but also replaces the blood already lost. Intramuscular blood injection is, however, an equally good hæmostatic, and requires a less skilful technique. Transfusion is also invaluable in the grave anæmias of infancy, such as occur in icterus gravis, congenital hæmolytic anæmia, and the more severe instances of anæmia in premature infants. In older infants Von Jaksch's anæmia and the grosser degrees of nutritional anæmia are benefited by transfusions. Hæmophilia, severe purpura, the anæmic crises of acholuric jaundice, and the acute hæmolytic anæmia of Lederer afford additional examples of blood diseases which are directly improved by transfusion. Much has been made of the value of blood transfusion in the wasting diseases of infancy, but a preliminary blood count should always be done, for it may be that the blood is concentrated with a red cell count up to or above 5,000,000 per c.mm., and the hæmoglobin above 100 per cent., and under these circumstances transfusion of whole blood is inadvisable ; in order to restore the blood volume, it would be wiser to transfuse plasma or serum. This also applies to the treatment of shock caused by extensive burns, for the loss of plasma from a widely burnt surface reduces the blood volume and causes the blood to become concentrated and viscous. The blood pressure falls, and the percentage of hæmoglobin rises. Steps to counter the shock may have to be taken at the same time as, or even before, the burnt area is dealt with, and this should be done by the transfusion of plasma in order to restore the blood volume as speedily as possible. Blood pressure readings and hæmoglobin estimations may be used to indicate when sufficient plasma has been given ; in severe cases the amount may have to exceed the rough rule used in blood transfusions of 10 c.c. per lb. of body weight.

Transfusion is not to be recommended in the treatment of localised acute infections such as pneumonia, peritonitis, or pyelitis, but it is of undoubted benefit in some cases of septicæmia.

Precautions. 1. The first precaution is that the blood of the donor and recipient must be compatible. This should always be tested by direct matching.

2. The amount of blood to be given must be in proportion

to the size of the child. A safe rule is to allow 10 c.c. of blood for every pound of body-weight—thus to a baby weighing 5 lbs. 50 c.c. of blood would be ample. This roughly corresponds to giving two pints to an adult.

3. The speed with which the transfusion is given must be carefully watched, for with a rapid transfusion there is a real danger of so embarrassing the right side of the heart as to cause heart failure. Allowing 10 c.c. of blood per lb., about twenty minutes should be taken in actually running in the blood, and so it follows that the smaller the child, the slower must be the rate of transfusion. This makes it impossible to employ direct transfusion from donor to recipient, and the donor's blood will need to be citrated by allowing 1 c.c. of a 2 per cent. solution of sodium citrate for every 10 c.c. of blood. Blood may also be given intravenously drop by drop, using the technique described for giving continuous intravenous saline (see p. 100).

In infancy, and often in older childhood, it is necessary to expose a vein by incision under local anæsthesia, and if this is to be done one has the choice of several sites. The internal saphenous vein as it crosses in front of the internal malleolus is often used, and has the advantage that it is as far as possible from the heart. Alternatively the median basilic vein may be chosen. The superior longitudinal sinus has been employed by direct puncture through the anterior fontanelle, but the risk of penetrating right through the sinus and injecting the blood into the subdural space, and the possibility of producing thrombosis of the sinus, prevent it from being customarily used.

CHAPTER XIX

DISEASES OF THE ENDOCRINE GLANDS

THE THYROID

THE secretion of the thyroid gland exerts a profound stimulus on the processes of metabolism, a stimulating influence which in childhood affects both physical growth and mental development. The active principle of the gland is an iodine-containing compound, and for the proper functioning of the thyroid an ingestion of minute amounts of iodine in the diet is necessary, otherwise the gland is liable to undergo a simple hypertrophy (goitre). The thyroid gland is the only member of the endocrines which, when given therapeutically by mouth, can be relied upon to exert an action, and in this connection it is of interest that the thyroid, by means of the thyroglossal duct, has a developmental connection with the alimentary tract. A deficient secretion, if dating from birth, leads to the clinical condition of cretinism, or if acquired later on gives rise to myxœdema, and excessive secretion leads to exophthalmic goitre.

Cretinism

Cretinism in this country only occurs in sporadic form, and the thyroid gland is either entirely absent or much undersized. Endemic cretinism is met with in parts of the world where goitre is common, and then may affect more than one member of a family. In the endemic cases the gland may be considerably enlarged, although its secretion is deficient.

The circumstances under which sporadic cretinism arises are not understood. Heredity plays no part. The condition is equally distributed between the sexes. Although the secretion of the thyroid is deficient or entirely absent from birth, the earliest manifestations do not appear until the infant is about twelve weeks old, and therefore the diagnosis cannot be made before that age. It would seem that the fœtus stores enough thyroid secretion derived from maternal sources to last for about three months.

Symptoms. When once the symptoms of cretinism have developed, the clinical picture is characteristic, and all cretins bear a remarkable similarity to each other. Although they appear plump enough, it is soon apparent that they are stunted in height.

The head is relatively broad and the features are coarse and pig-like. The eyelids are heavy and the palpebral fissures narrowed, the nose is squat, the lips are thickened, and the tongue is enlarged and protruding and its papillæ stand out prominently; indeed it may become so hypertrophied as to be literally too large for the mouth and may give rise to obstruction to breathing and difficulty in sucking and swallowing. The complexion is sallow because of anæmia, the hair is scanty, dry, and brittle, and the eyebrows are thinned. The skin is dry; the subcutaneous tissues become infiltrated with a solid œdema, supraclavicular pads of fat develop and form a collar round the base of the neck, the limbs are thickened, and the fingers are broad and stumpy. The abdomen is protuberant, and frequently an umbilical hernia is present. The circulation is sluggish, the extremities are cold, and the temperature is subnormal. Physical development is retarded, the teeth erupt late and often irregularly, and the anterior fontanelle may remain open into the third year. By this time, although the cretin maintains a squat solid figure, the weight and height are both much below the average for the age. Not only is the growth of the skeleton delayed, but calcification of the epiphyses proceeds irregularly, and this is often demonstrable in X-rays of the long bones.

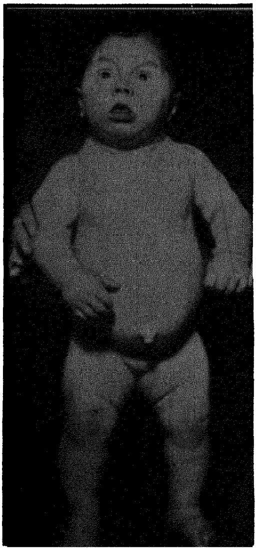

FIG. 69. Untreated cretin aged four years.

In many cases it is impossible to palpate either the lateral lobes or the isthmus of the thyroid.

The retardation of mental development is as striking as the peculiar physique. The expression is one of downright dullness, and the reaction time is noticeably slow. The infant loses interest in the goings-on around, and it may be difficult to rouse him to feed. Smiling and crying are infrequent and require an unusual amount of provocation, but when in full cry the forehead becomes much puckered and the voice is strangely hoarse and croaking. Constipation is a most constant feature, and is severe. If the condition remains untreated, development is very slow, walking and talking are much delayed, and at the age of puberty an untreated cretin has the mental equipment of a child of only two or three years. Secondary sexual characters do not develop.

At post-mortem examination the thyroid gland may be entirely absent; in others only the atrophied remnant of the gland can be found.

Diagnosis. When a cretin has once been seen, others should be recognised at a glance, for they are all alike. When the typical characteristics are beginning to unfold at about three months of age, doubt may at first be entertained, and it would then be as well to wait two or three weeks before beginning treatment in order to let the picture become more complete. The rapid alteration in the infant's appearance after a few weeks of treatment by thyroid will also confirm the diagnosis, if that were needed. The only condition with which cretinism is at all likely to be confused is mongolism, although in actual fact the two conditions are quite dissimilar, and mistakes should not occur when once a typical example of each has been seen. The differences between the cretin and the mongol are set out on p. 589.

Prognosis. There can hardly be any condition in which the prognosis is more favourably affected by early diagnosis and persistent treatment. When treatment begins really early—at the fourth month or so—both physical and mental development go ahead rapidly. The improvement in the infant's appearance is such that within a few months it is impossible to recognise the baby as a cretin, indeed a change can be detected after three or four weeks, and from then on growth proceeds normally, and eventually at puberty the usual sexual changes take place. The growth in height is at first particularly rapid, and it is therefore important to see that the usual safeguards against rickets are being carried out. With early treatment mental growth may be as successful as physical growth, but unhappily this is not always the case, and a degree of simple-mindedness may remain. When treatment is delayed until the end of the first year mental growth is seldom as satisfactory as physical growth, and the child is likely to remain backward, with an intelligence quotient three or four years behind the average. When treatment is not commenced until five years of age very little can be hoped for, although some amelioration of the physical appearance may be brought about.

The amount of improvement that can take place may be illustrated by three cases. One, an undoubted cretin at three months of age, was under the writer's care and won first prize at a baby show when a year old; another, recorded by Cockayne, won a scholarship at school; a third, recorded by Thomson, eventually married and raised a healthy family.

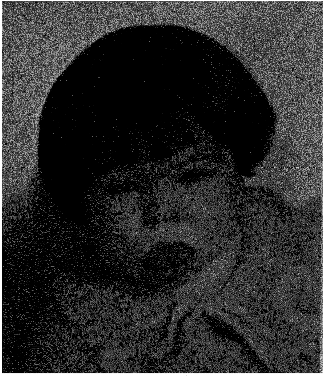

FIG. 70. Untreated cretin, aged eighteen months.

Treatment needs to be kept up throughout life. If the administration of thyroid stops, the child gradually sinks back into a myxœdematous state, although improvement takes place when treatment is re-started.

Treatment. The essence of treatment is to supply the child with thyroid, and this should be given by mouth in the form of thyroideum siccum. It is wise to start with a small dose and gradually increase this, being careful not to overstep the child's limit of tolerance. For an infant gr. $\frac{1}{5}$ twice a day should be the initial dose, the amount being raised by gr. $\frac{1}{5}$ after a fortnight. In infancy a total daily dose of one grain of dried thyroid will usually be sufficient, but in older children as much as five grains a day may be required. The regulation of the dose must be gauged by clinical standards,[1] since the amount required varies with each individual and cannot be assessed merely by the age. Overdosage leads at first to irritability, disturbed sleep, and a rapid pulse, followed by tremors, loss of weight, and diarrhœa, and indicates the need for a temporary reduction in the dose. On the other hand, persistent constipation may be taken as an indication for more thyroid, in fact the state of the bowels, whether constipated or loose, is a most useful indication of whether too little or too much of the gland is being given.

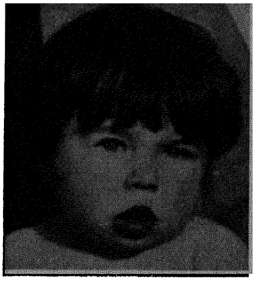

FIG. 71. The same child as in Fig. 70, after two months of treatment with thyroid.

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[1] Recent studies have shown that hypothyroidism is usually accompanied by an increase in the serum cholesterol, which falls to normal under treatment, and it has been suggested that estimations of the cholesterol may afford an indication of the efficiency of treatment.

Myxœdema

In this condition the thyroid function becomes defective at some time after birth. Should this occur early there may be difficulty in distinguishing the condition from cretinism, but as a rule the history is that the child has developed normally for some years, and then, perhaps following some acute infection, a slow change for the worse has set in. The child becomes lethargic, dull, dirty, the hair falls, the skin becomes dry, the subcutaneous tissues thicken, the features coarsen, the voice becomes hoarse, and growth slows down.

The resemblance to cretinism in a severe case renders the diagnosis easy, and in milder cases the many small points enumerated above should suggest the condition.

Treatment consists of supplying sufficient thyroid to enable the child to return to, and maintain, a normal condition.

Exophthalmic Goitre

This is a rare condition in childhood. An instance of the disease has been recorded in a newborn infant whose mother was suffering from the condition, and a few cases have been reported in the early years, but the majority arise during the later years of childhood.

The symptoms are much the same as in adults. The gland becomes enlarged, exophthalmos is nearly always present in company with von Graefe's and Stellwag's signs, and there is also tachycardia. The loss of weight may be severe, sweating and a tendency to diarrhœa are common, and the basal metabolic rate is increased. Tremor is not so frequent a symptom as in adults, but it may take a more coarse or choreic form. Emotionalism is very noticeable, and is shown by rapid changes from perversity to exhilaration.

The prognosis in childhood is more favourable, and surgical interference is not as a rule required. Treatment consists first and foremost of complete rest in bed under the charge of a trained and understanding nurse. The diet must be nutritious, and malted preparations are a useful addition. Iodine (as Lugol's solution, three to five minims three times a day) should be prescribed. In more severe cases carefully controlled X-ray exposure over the gland may be employed. A partial thyroidectomy should only be considered in children when a thorough trial of the measures given above has proved fruitless.

Goitre

Simple enlargement of the thyroid gland occurs endemically in certain parts of the world, and in this country in Derbyshire. Sporadic cases also arise in children, as in adults. The thyroid may enlarge before birth and cause difficulty in delivery. In infancy the most important symptom is breathlessness, due to compression of the trachea ; the dyspnœa may be urgent, giving rise to stridor and causing a degree of asphyxia.

Goitre is also not infrequent at the time of puberty, and is then more common in girls than boys and seems particularly to affect tall children. The condition subsides as puberty passes, but its disappearance can be hastened by giving small doses of iodine, such as three minims of Lugol's solution thrice daily.

The cause of the enlargement of the thyroid in sporadic cases is not understood. McCarrison has shown that where the condition is endemic the cause may lie in a bacterially contaminated water supply, and prevention lies in boiling and filtering the drinking water. Soils deficient in their iodine content are also associated with endemic goitre.

Acute Thyroiditis

Acute inflammation of the thyroid is rare in children. It may complicate the acute specific fevers or be secondary to sepsis elsewhere in the body. The gland becomes acutely swollen and tender, swallowing is painful, and dyspnœa may be severe.

Hot fomentations should be applied over the gland. The inflammation may subside in a few days or may go on to suppuration and call for surgical relief.

THE PARATHYROIDS

Evidence of disease of the parathyroids in children is very rare. The experimental extirpation of these glands leads to severe tetany, and in human beings this has occasionally followed operations on the thyroid. Hypoparathyroidism is not met with in childhood.

Hyperparathyroidism[1] (Osteitis Fibrosa Generalisata) has been recorded in childhood. The condition is due to an adenoma of the parathyroids, and the effect is to drain calcium from the bones, causing a marked rise in the blood calcium and an increased excretion of calcium in the stools and urine. The bones become tender, fractures and bending occur readily, and X-ray examination shows rarefaction of the bones, the appearance of cystic spaces in the cancellous tissue, and a soft fibrous compact-bone. Hypotonia, vomiting, and muscular cramps may occur.

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[1] Hunter, Donald, *Brit. Jour. Surg.*, 1931–32, **19**, 203.

Treatment consists of the surgical removal of the parathyroid tumour.

THE PITUITARY GLAND

The study of the pituitary gland is rendered difficult for three reasons. Firstly, the gland is composed of two distinct portions (1) a pars anterior, and (2) a pars intermedia and pars nervosa ; these two portions possess different functions, and clinically we may have to recognise various combinations of overaction and underaction produced by the different parts of the gland in one and the same patient. Secondly the gland is situated in the bony sella turcica at the base of the brain, and tumours of one part of the gland are very likely to interfere with the function of the other parts by direct pressure, and also with the neighbouring parts of the brain. Thirdly the pituitary has been well called the master gland of the endocrines, as it exerts a profound effect on the other members, stimulating the thyroid, adrenals, thymus and gonads, and depressing the pancreas. Therefore in lesions of the pituitary secondary effects may arise from interference with the functions of other glands.

The *Pars Anterior* contains chromophil (eosinophil) and chromophobe cells. The chromophil cells produce a hormone which regulates skeletal and visceral growth and stimulates the development and function of the gonads.

The *Pars Intermedia* is responsible for the secretion of pituitrin, which passes *viâ* the pars nervosa to the hypothalamic region of the brain and so to the third ventricle. Some of the symptoms usually associated with disordered action of the pituitary, such as obesity, and the polyuria of diabetes insipidus, are the outcome of interference with hypothalamic function.

We may now consider the various forms which pituitary disease may take.

Gigantism

Overaction of the chromophil cells of the pars anterior is less common in children than in adults, in whom it produces acromegaly. In childhood gigantism results, the effect being a simple overgrowth of the whole child, to which the features of acromegaly may be added as adult life is reached. Glycosuria has been noted in some instances, and may be followed at a later stage by an increased tolerance for sugar. Rarely acromegalic characteristics—large extremities, coarse features with relative overgrowth of the skull and jaw and prominent supraorbital ridges, and kyphosis—have been recorded in children.

In some cases the condition is brought about by a chromophil adenoma in the anterior lobe, and to the signs of overgrowth may be added such local signs of a pituitary tumour as bitemporal hemianopia, and there may be X-ray evidence of an expanded sella turcica. Occasionally gigantism has occurred with hypoplasia of the genitalia, due to a mixed tumour of chromophil and chromophobe cells.

When the signs of a tumour are associated with failing vision, operation on the pituitary is justifiable. Otherwise deep X-ray treatment may be tried.

Basophil (Chromophobe) Adenoma

A syndrome caused by small basophil adenomata has been described by Cushing. The symptoms comprise a rapid and painful obesity with striæ atrophicæ in the skin, generalised hirsuties, high blood pressure, softening of the bones, and cyanotic discoloration of the extremities. The tumours are too small to produce local pressure effects on the brain. Treatment by X-ray exposures should be given.

REFERENCES

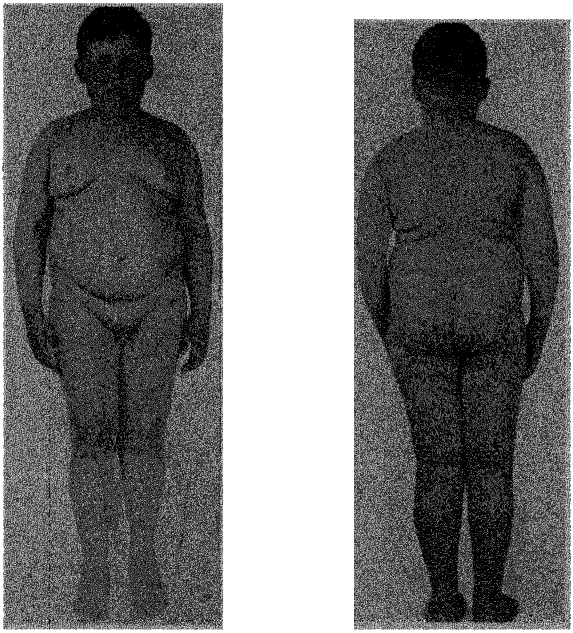
CUSHING, H. *Bull. Johns Hopkins Hosp.*, 1932, **1**, 187.
DOTT, N. U., and BAILEY, P. *Brit. Jour. Surg.*, 1925, **13**.

Deficient Function of the Pars Anterior

This is a clinical rarity, of which the chief symptom is infantilism. The two varieties—progeria and Simmond's disease—have close similarities. In progeria there is from an early age remarkable stunting of stature, with delayed closure of the skull, loss of hair and of subcutaneous fat so that the skin is wrinkled, and arteriosclerosis. The appearance is one of premature senility. Simmond's disease usually follows some acute infection, and is characterised by a sudden arrest of growth, with premature fusion of the epiphyses, loss of subcutaneous fat and wrinkling of the skin, and absence of pubertal changes. The intelligence is not affected. The condition is due to infarction of the artery to the pituitary body, with cutting off of the blood supply to the pars anterior.

Dystrophia Adiposo-genitalis (Frohlich's Syndrome)

This condition is the result of interference with the function of the posterior lobe, and often of the hypothalamus as well. It may come about as a result of basal meningitis—usually syphilitic—or may follow encephalitis or fractures at the base of the skull which have led to local hæmorrhage, but is usually due to a slow-growing tumour in the suprapituitary region or to a cyst of Rathke's pouch. In this last group the tumour or cyst may gradually undergo calcification, so that after other symptoms have been present for some years an X-ray may show specks of calcification above the pituitary fossa, and there may also appear some distortion of the sella with erosion of the clinoid processes, but these changes are seldom apparent before puberty.



FIGS. 72 and 73. Dystrophia adiposo-genitalis (Frohlich's syndrome) in a boy aged eleven years.

The chief symptom is obesity, the fat being laid down somewhat after the feminine distribution, the breasts, buttocks, and lower abdomen being particularly adipose. There is usually some stunting of stature with delay in ossification, which may be demonstrated by X-raying the carpal centres. The external genitalia are immature, and in a boy the penis is strikingly diminutive and sunken in the surrounding fat, while the testicles may be undescended. The hair on the scalp is often fine and silky, but hair fails to appear in the axillæ or on the pubes or limbs. By comparison with the trunk, the hands are often small and the fingers long and tapering. As a rule the sugar tolerance is increased. The mentality is sometimes unimpaired, but more often there is a varying degree of turpitude, particularly in cases due to a tumour. After some years such local signs of a tumour as bilateral temporal hemianopia or optic atrophy may appear.

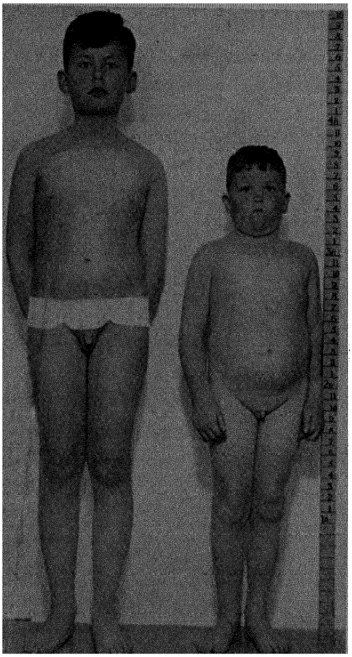


FIG. 74. Pituitary dwarfism, illustrated by the boy on the right, who is not only short, but also obese, and has undescended testes. The boy on the left is normal. Both are aged nine years.

The label of "pituitary obesity" is too often applied to fat children, the majority of whom owe their obesity to overeating and insufficient exercise rather than to any definite endocrine abnormality. As a rule children with pituitary obesity have a really small appetite, and in addition the genital hypoplasia is a further characteristic distinguishing them from overfed children.

Treatment. The obesity can to some extent be controlled by diet and drugs, although any striking loss of weight is not to be expected, and in this respect the results of treatment do not compare with those obtained in children whose obesity is exogenous rather than endogenous. The diet should be one in which starches, especially bread, potatoes, root vegetables, and cereals, are withheld (see p. 171). Of drugs, whole pituitary gland may be given by mouth (B. W. & Co. tablets, from gr. 1 to 5 ter die), and may be combined with smaller doses (gr. $\frac{1}{8}$ to $\frac{1}{2}$) of dried thyroid. The results of treatment are, however, often disappointing. Occasionally a simple tumour may be dealt with surgically with good results, or X-ray treatment may be employed. The possibility of syphilis as a cause must be borne in mind.

Two further varieties of pituitary obesity must be mentioned. In one (the Schüller-Christian syndrome) there is a primary fault in lipoid metabolism, and xanthomatous cells invade the pituitary and the bones of the skull. The condition is more fully described under the lipoid disorders (see p. 167). In the other variety (Lawrence-Moon-Biedl syndrome) the condition of dystrophia adiposo-genitalis is associated with retinitis pigmentosa, polydactyly, and mental defect. The disease is a familial one.

Diabetes Insipidus

This condition is characterised by severe thirst and the passage of enormous amounts of very pale urine of low specific gravity. There is no glycosuria. The lesion is situated in the hypothalamus, but may be accompanied by evidence of a disordered pituitary function. In children syphilitic basal meningitis is the usual cause, but it may follow encephalitis or trauma to the base of the skull. The symptoms can be relieved by the daily injection of 1 c.c. of pituitrin subcutaneously. Anti-syphilitic treatment should also be given in appropriate cases.

THE PINEAL GLAND

The pineal gland is generally held to control the development of the sex glands, which develop at puberty coincidentally with involution of the pineal.

Tumours of the pineal body are almost confined to boys, in whom they cause a precocious enlargement of the genitalia with the hirsuties characteristic of puberty. The mentality may also be forward. The precocious puberty may make its appearance at any age after about the fourth year. The tumour is most commonly a teratoma ; in addition to precocity it may give rise to such localising signs of its presence as paralysis of upward and downward movement of the eyes, diplopia, and later on to signs of increasing intracranial tension.

Treatment is ineffective, and death comes about at an early age.

THE SUPRARENAL GLANDS

The suprarenal glands consist of two portions, a cortex and a medulla, which differ in their embryological development and possess separate functions. The medullary portion is composed of chromaffin tissue and is responsible for the production of adrenalin, while the cortex influences growth and sexual characteristics.

Suprarenal Hæmorrhage

Hæmorrhage into the suprarenals of the newborn may be due to trauma during labour, or to asphyxia, or to too vigorous attempts at artificial respiration. Of cases that arise after birth, the majority occur within the first few months, and are then generally secondary to acute infections—the author recalls instances of meningococcal infection, and Vincent's infection, as immediate forerunners—but the cause is sometimes obscure. The hæmorrhage may be unilateral (usually the right side) or bilateral, and may cause a sufficiently large swelling to be felt during life. Rupture of the swelling into the peritoneal cavity has been recorded.

The onset is rapid. The infant becomes drowsy, the pulse rises and may become uncountable, respirations become shallow and frequent, slight diarrhœa is usual, and a dusky mottling develops in the skin. Convulsions may occur, and the infant dies. The whole disease may only last a few hours. Purpuric hæmorrhages often appear a few hours before death.

Although the disease is, as a rule, rapidly fatal, the following case suggests that at least a temporary recovery may be made. A female infant born at full term, and after a normal labour, weighed 7½ lbs. She was seen at ten weeks of age and then weighed only 5½ lbs. In spite of careful treatment, the baby steadily wasted and died when three months old. At autopsy the only pathological finding lay in the right suprarenal, which was distended with an old and decolourised blood-clot.

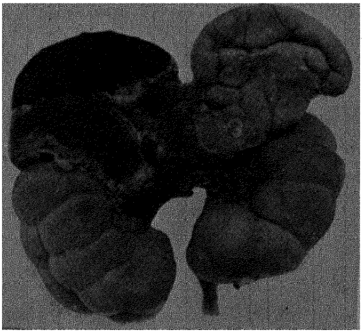


FIG. 75. Suprarenal hæmorrhage in an infant aged nineteen days.

As a rule the opportunity for treatment does not arise, but Goldzicher and Greenwald[1] have recorded a case which recovered after repeated injections of an extract of suprarenal cortex.

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[1] Goldzicher and Greenwald, *Amer. Jour. Dis. Child.*, 1928, **36**, 324.

Addison's Disease

This is a rare condition in childhood, and is scarcely ever met with before the end of the first decade. The symptoms are identical with those in adults, and comprise progressive asthenia and wasting, a low blood pressure, and a brown pigmentation of the skin and mucous membranes. The condition is generally fatal within a year or two.

Nearly always the disease is the result of caseous tuberculosis of one or both suprarenals, the infection reaching the glands from the intestine, peritoneum, or lungs. Similar symptoms have been recorded from absence or simple atrophy of the glands.

Treatment is unsatisfactory, death usually occurring within a year or two of the onset. It is now known that many of the symptoms are dependent on a loss of sodium from the body, and considerable relief may follow the oral administration of common salt in doses of from 3 to 10 grammes daily. Encouraging results [1] have recently been reported to follow repeated injections of a cortical extract.

New Growths

New growths of the suprarenal must be looked upon as rarities in children. There are two main types—(1) malignant neuroblastoma arising from the medullary portion of the gland, and (2) sarcoma of the suprarenal cortex. The former is the more common.

Medullary Neuroblastoma

These tumours are subdivided into two types—Hutchison's tumour and Pepper's tumour—according to the distribution of the secondary deposits.

Hutchison's Tumour has been reported in children as old as six years, but is usually met with under three years of age. Boys are affected a little more often than girls. The early symptoms are vague, and the condition is only suspected when the characteristic secondary deposits arise. These occur in the orbits, on the flat bones of the skull, and on the inner side of the ribs. Hæmorrhage into the eyelids and proptosis are often the first evidence of these deposits, and as a rule the eye first to be affected is on the same side as the diseased suprarenal. Multiple tumours on the skull soon follow. Those on the outer surface are hard and may be as large as a hen's egg, while those on the inner surface give rise to signs of an intracranial neoplasm with headache and optic neuritis. As a rule by the time these secondary deposits have appeared the primary tumour in the abdomen can also be felt, but this is not always so, in fact the abdominal tumour sometimes remains impalpable until death. The lymphatic glands above the inner end of the clavicle on the side of the affected suprarenal are usually enlarged.

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[1] Levy Simpson, *Quart. Jour. Med.*, 1932, New Series I., 99.

The condition must be distinguished from chloroma—in which tumours also form in the orbit and cause proptosis and hæmorrhage into the eyelids—by the leukæmic blood picture which is characteristic of chloroma. The blood picture in cases of Hutchison's tumour shows little beyond a progressive secondary anæmia. Orbital hæmorrhages may also occur in scurvy, but the evidence of hæmorrhage into the gums and beneath the periosteum of the long bones, the presence of blood cells in the urine, and the scorbutic beading of the ribs will serve to distinguish this condition.

Treatment is of no avail, and the disease is fatal within six months.

Pepper's Tumour. This usually affects the right suprarenal, and gives rise to a diffuse secondary invasion of the liver. The condition arises soon after birth, and is fatal before six months of age.

The first symptom is a progressive enlargement of the liver, noticed soon after birth. The whole organ enlarges evenly, and is firm and smooth. There is neither jaundice nor ascites and the spleen is not felt. The abdomen may become considerably distended, and the umbilicus becomes unfolded. At postmortem examination the whole liver is diffusely invaded by growth.

Tumour of the Suprarenal Cortex

This is a rare condition, and affects girls much more often than boys. Symptoms may appear at any age, and differ according to the sex of the child. Lightwood has recorded an instance in a male infant aged eighteen weeks. In girls the body takes on the features of masculinity, hair grows on the face, trunk, and arms, the clitoris enlarges, but menstruation is absent. Adiposity is a prominent symptom, the cheeks become bloated and often ruddy, and there may be great muscular development. In boys the secondary sexual characters appear prematurely, the penis enlarges, pubic hair grows thickly, and there may be a prodigious muscular development (so-called Infant Hercules). The symptoms are very similar to those of a pineal tumour.

Tumours of the suprarenal cortex are slow-growing and show no early tendency to metastasise. A regression of symptoms has followed upon surgical removal of the tumour.

A similar condition of virilism, enlargement of the genitalia, and hirsuties, may be due to hyperplasia of the supra-renal cortex affecting one or both glands. There is some evidence to suppose that the overaction of the suprarenal cortex in these cases depends upon a hypersecretion of the basophil cells of the anterior lobe of the pituitary gland. Similar symptoms have also been recorded in patients who have died from a malignant tumour of the thymus gland.[1] Tumours of the suprarenal cortex, and cortical hyperplasia, give rise to a considerable increase in the urinary excretion of ketosteroids—substances normally produced by the suprarenal cortex.

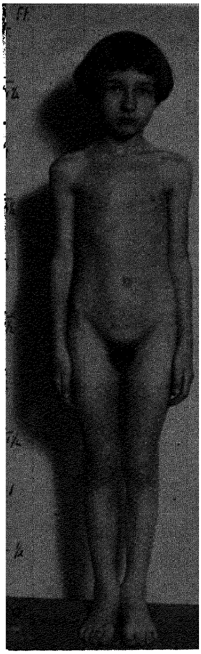

FIG. 76. Precocious pubic hirsuties in a girl aged six years. The clitoris was enlarged, but there was no menstruation. The cause probably lay in hyperplasia of the suprarenal cortex.

THE GONADS

The function of the ovaries and testes may err in the direction of over- or undersecretion.

Hypersecretion leads to precocious puberty, indicated in girls by premature development of the breasts, the appearance of pubic and axillary hair, and an early onset of menstruation, and in boys by the premature appearance of hair on the face, chest, axillæ and pubes, and enlargement of the genitalia. The condition is met with more frequently in girls than in boys. In both sexes there may be initially a rapid growth in height, but this soon comes to an end owing to early union of the epiphyses. When the symptoms are associated with a tumour of the ovary or testis, its removal may be followed by a gradual disappearance of the symptoms.

Hyposecretion, or eunuchoidism, is more common in boys than in girls, and in the former is often associated with incomplete descent of the testes into the scrotum. If the testes can be felt, they are noticeably small and devoid of sensation. In both sexes the children are tall and somewhat obese, the union of the epiphyses is delayed, and in boys the body may assume a feminine contour. Puberty is delayed, maybe indefinitely, and the trunk remains hairless. The mentality is not affected.

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[1] Leyton, O., *Lancet*, 1934, *i.*, 1221.

Undescended Testicles. Medical opinion is often sought because a boy has one, or both, of his testicles undescended. It must be remembered that in a young child the testicle can be retracted well above the scrotum, appearing as though undescended, but gentle manipulation may cause the testicle to regain the scrotum, thus disproving any abnormality. Mention has already been made of undescended testicles in Frohlich's syndrome and in eunuchoidism. Failure of descent is sometimes associated with an inguinal hernia, or may be due to the testicle occupying an ectopic situation, usually superficial to the inguinal canal ; in either of these circumstances spontaneous descent will not occur, treatment by hormones is contraindicated, and operation should be advised. Of the remaining cases, the majority undergo spontaneous descent before or during puberty, and treatment should be deferred until it can be seen whether such descent takes place. If this does not occur, operation is generally necessary. Hormone treatment consists of giving intramuscular injections of anterior pituitary preparations such as Prolan ar Pregnyl ; these usually lead to swelling of the testicles, but only occasionally bring about descent, and they should not be given before puberty. If surgical treatment is subsequently necessary, hormone therapy may render operation easier.

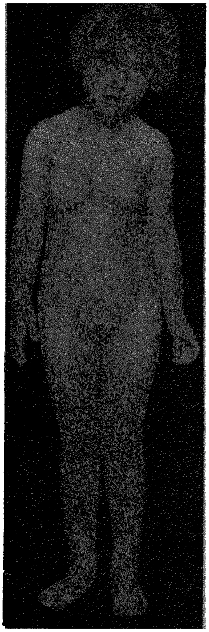

FIG. 77. Precocious puberty in a girl aged three years and eleven months. Menstruation had already commenced.

THE THYMUS

The function of the thymus is but imperfectly understood, although it is thought to exert an inhibiting influence on the development of the sex glands. Certainly the gland atrophies as puberty approaches, being eventually replaced by fat and fibrous tissue. The organ is largest at birth. Figures obtained from the post-mortem room at the Hospital for Sick Children showed that from birth until four years the average weight of the thymus was 5·3 gms., the extremes being 2 gms. and 12 gms. These figures exclude those cases in which death took place suddenly and unexpectedly, and in which an obviously enlarged thymus was found at autopsy. In such cases the thymus may weigh up to 50 gms.—ten times the normal weight.

The two chief clinical conditions which arise from enlarged thymus are thymic asthma and status lymphaticus (lymphatism).

Thymic Asthma (Kop's Asthma)

This is a rare condition which occurs in young infants, and is characterised by attacks of dyspnœa with inspiratory and expiratory stridor, and cyanosis. Others may suffer from sudden syncopal attacks, and these may recur during the first two or three years. In these attacks the child may seem to faint, falls down unconscious, and becomes cold and blue. There may be convulsive movements, and death may result, although as a rule recovery is made after a few minutes. A history sometimes obtained in young infants, and which should arouse suspicion of a large thymus, is that the mother fears to bath her baby because immersion in water brings on a syncopal attack.

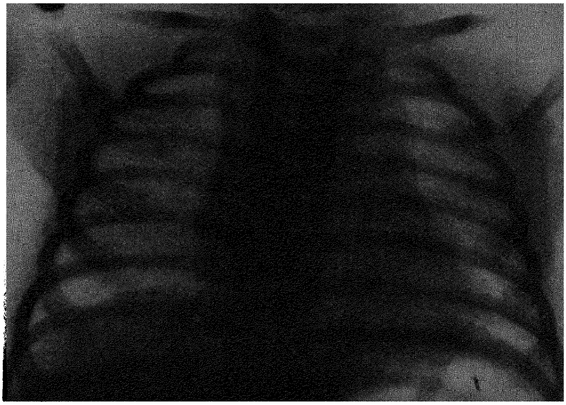

FIG. 78. X-ray showing the shadow caused by a large thymus. From an infant ten days old who had attacks of cyanosis with stridor. The symptoms cleared up and the shadow disappeared following radium treatment over the thymus.

If the thymic enlargement is detectable clinically, it will be revealed by dullness to percussion over the manubrium sterni and for about a finger-breadth to each side, and radiological examination shows a broad shadow in the midline above the heart shadow. A lateral X-ray view may also show the trachea compressed or thrust backward by the large gland. Rarely the thymus may be felt projecting in the episternal notch.

A relief of symptoms may be brought about by giving one or two exposures of the upper part of the chest to deep X-rays, or by the application of radium over the thymus.

Status Lymphaticus

The post-mortem findings in this condition consist of gross enlargement of the thymus, with hypertrophy of the spleen and a general increase of the lymphatic tissues. The thymus may be so extensive that its three lobes, which normally just reach to the base of the heart, cover the anterior surface of the pericardium and completely obscure the heart. Its weight may be increased up to 30 or 50 gms.

Certain peculiar clinical states may arise in children in whom these hyperplastic lymphatic changes are found at autopsy. The most disastrous is a sudden death for which there seems no adequate cause. It may occur as a result of some quite mild infection, particularly of the respiratory passages. Thus a child of seven years died unexpectedly during a mild attack of bronchitis, and another case was that of an infant of ten months who died in the same way. At autopsy no adequate cause of death could be found in either of these children, but the thymus was very enlarged, weighing 37 gms. in the older child and 50 gms. in the younger. Anæsthesia presents a great risk to these children, many of the deaths under anæsthesia being associated with the typical findings of lymphatism at autopsy.

The diagnosis of lymphatism is seldom made until serious symptoms have occurred. The children are for the most part plump and flabby, although so are many others who do not exhibit lymphatism. If there is reason to suspect the presence of lymphatism, the child should be protected as much as possible from infections, and surgical operations should only be undertaken if emergencies arise making them essential, and the risk should be explained to the parents. Treatment of the thymus by deep X-rays or radium should be undertaken with the object of causing the gland to atrophy.

CHAPTER XX

DISEASES OF THE NERVOUS SYSTEM

ACUTE SUPPURATIVE MENINGITIS

THE organisms most commonly responsible for acute suppurative meningitis are the streptococcus and the pneumococcus, and less commonly the staphylococcus or the influenza bacillus. The forms of meningitis produced by these organisms have so much in common that they may conveniently be considered together.

Almost always acute suppurative meningitis occurs secondarily to infection elsewhere. In pneumococcal and streptococcal cases the most frequent forerunners are otitis media and mastoiditis or pneumonia, while staphylococcal meningitis is generally secondary to infections of the skin or bones. Occasionally the pneumococcal variety seems to arise *de novo*, but even in such cases it is probable that the organism has been lurking symptomless in the throat or elsewhere. Influenzal meningitis is most frequently met with under two years of age, in fact, of 220 cases collected by Rivers [1] no less than 152 occurred during the first two years.

Symptoms. The onset is generally sudden and may be heralded by convulsions or vomiting. At the outset the child is likely to be irritable, and if old enough will complain of intense headache, but in other cases the onset may be masked by the symptoms of the primary illness. Fever is likely to be high, up to 104° F. As a rule the classical signs of meningitis develop quickly. The fontanelle is tense and bulging, stiffness of the neck muscles soon appears, and is followed shortly by retraction of the head and a positive Kernig's sign. The diagnosis is confirmed by examination of the cerebro-spinal fluid, which is under increased pressure, cloudy, with a high count of polymorphonuclear cells ; the protein is increased, the chlorides are as a rule slightly diminished, and the sugar is either diminished or absent. The causal organism may be found by examination of a direct smear, or by culturing the fluid.

The illness runs a rapid course, seldom lasting more than a week. Irritability soon gives way to drowsiness and coma, and there may be frequent convulsive attacks just before death. The course of influenzal meningitis is usually a little longer, lasting perhaps for two or three weeks. At post-mortem examination the surface of the brain and spinal cord is smeared with a purulent yellow exudate which may be so dense as to hide the swollen and engorged meningeal veins. In cases arising secondarily to ear disease the meningitis may be localised to the neighbourhood of the infected mastoid.

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[1] Rivers, T. N., *Amer. Jour. Dis. Child.*, 1922, **24**, 102.

Before the introduction of the sulphanilamide group of drugs, the prognosis was thoroughly bad, the mortality being in the neighbourhood of 95 per cent. Isolated instances of recovery were recorded from time to time and were usually pneumococcal, although it is probable that recovery was due as much to the slightness of the infection as to the variety of therapy employed. With modern chemotherapy the chance of recovery has been enhanced, but the illness remains an exceedingly grave one, and carries a high mortality.

Treatment. The most important aspect of treatment lies in chemotherapy with the sulphanilamide group of drugs, which should be given in full doses (see p. 715). Oral administration is generally sufficient, but should vomiting or coma make this route impracticable, a soluble preparation suitable for intramuscular use should be selected. Care must then be taken to see that the injection is not given in the immediate vicinity of any important nerve, as instances of nerve degeneration with permanent paralysis have followed the incautious injection of these drugs. It is doubtful whether repeated lumbar puncture has any curative value, although it may be carried out in the face of a rising intracranial pressure, as would be denoted by severe headache, frequent vomiting, or deepening coma.

When suppurative meningitis arises secondarily to ear disease, surgery may be required, and co-operation with an aural surgeon is then essential.

MENINGOCOCCAL MENINGITIS (POSTERIOR BASIC MENINGITIS : CEREBRO-SPINAL MENINGITIS)

Although it is customary to divide meningococcal meningitis in children into two groups, the one affecting infants, occurring sporadically, and called posterior basic meningitis, the other chiefly affecting older children, occurring both sporadically and epidemically, and called cerebro-spinal meningitis, both diseases are due to infection by the meningococcus, and on bacteriological grounds cannot be distinguished. The treatment of the two forms is the same, but the disease in infancy presents clinical features which warrant a separate description.

Cerebro-spinal meningitis may occur at any age. In this country it is generally sporadic, but may assume epidemic proportions.

Symptoms. The onset is generally sudden, and is characterised by vomiting, headache, sometimes pain in the back, and a temperature up to 102° to 104° F. In young children there may be one or more convulsions at the onset. From the beginning of the illness the signs of meningitis are generally evident; the neck muscles are so stiff that the whole child can be raised by lifting the head off the pillow, and the stiffness steadily increases until the head is held retracted. Kernig's sign and Brudzinski's sign (which consists of flexion of the hips when the head is raised) are positive. At first there may be delirium, which gives place after a few days to drowsiness and coma. Herpes may be present on the lips, and there may be a fine purpuric rash scattered on the trunk, which has given the name "spotted fever" to the disease An internal strabismus may develop, and blindness is an occasional symptom and is evidently of central origin, for examination of the fundus oculi shows no changes. When recovery takes place the blindness generally passes off completely, but permanent loss of sight may remain. Panophthalmitis may also occur, but is fortunately a rare complication; it arises as a metastatic abscess, and almost always results in destruction of the eyeball. Deafness is also a complication which may be permanent.

The *posterior basic* variety is confined to infants, occurs sporadically, and has its maximum incidence in the spring. In a typical case the onset is brisk, with fever, vomiting, and convulsions. Head-retraction comes on quickly and may become so severe that the head is drawn back between the shoulders. The trunk and limbs are rigid, with the legs extended and the arms flexed. The tendon reflexes are at first brisk, and Kernig's sign and Brudzinski's sign are positive. The fontanelle is bulging and tense and loses its pulsation, and the pulse is rapid and at first full. A tâche cerebrale can often be elicited by lightly stroking the skin. The infant is often very sensitive to gentle stimuli, and may twitch at sudden sounds or even when touched. Blindness, of central origin with a normal appearance of the fundi, may develop, and the infant may exhibit coma-vigil, lying for long periods with open staring eyes, in which state a purulent conjunctivitis is likely to occur, and makes frequent cleansing of the eyes necessary. In the early stages the baby may give frequent piercing cries, but after a day or two lapses into coma. Squints are not uncommon, but herpes and purpuric rashes are not seen. A meningococcal arthritis is an occasional complication.

While the above description characterises a typical case, atypical varieties may be met with, and they often make the diagnosis a most difficult matter. For instance, the vomiting at the onset may be accompanied by such severe diarrhœa as to lead to a mistaken diagnosis of gastro-enteritis, and this is particularly likely to happen when the head-retraction and other signs of meningitis do not appear until several days have elapsed, while the fontanelle instead of bulging may be much depressed owing to the loss of fluid in the stools. The temperature is generally high, but occasionally may fall to subnormal within a day or two of the onset and may remain low for the remainder of the disease. A heavy glycosuria at the onset may also be misleading.

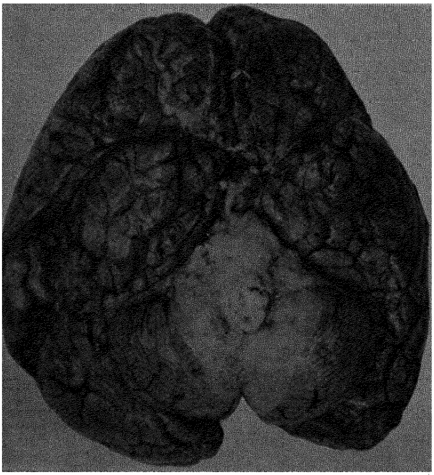

FIG. 79. The brain of a child aged 2½ years, who died of post-basic meningitis, showing the distribution of the exudate over the posterior third of the brain.

Pathology. Post-mortem examination shows the meninges covered with a yellow fibrino-purulent exudate, which in the posterior basic variety is situated particularly round the base of the brain in the posterior and middle fossæ, where it is likely to obliterate the foramina of Magendie and Luschka through which the cerebro-spinal fluid drains from the fourth ventricle to the cisterna magna, and so may cause hydrocephalus. The exudate may also spread over the vertex of the brain and down the spinal cord, in fact this is usually the case in the cerebro-spinal type. The meningeal vessels are congested and engorged and may occasionally become thrombosed before death.

Diagnosis. In a typical case with an illness of sudden onset, marked by fever, vomiting, possibly convulsions, and showing head-retraction and Kernig's sign, the diagnosis should present no difficulty; but milder cases occur in which the signs of meningitis may at first be poorly marked. Successful treatment depends very much upon early diagnosis, and therefore if the question of meningococcal meningitis arises a lumbar puncture should be performed forthwith. The changes in both cerebro-spinal and posterior basic types are identical. The fluid is under increased pressure, turbid and yellowish, and during the acute stage there is a polymorphonuclear cellular reaction. The chlorides are reduced, the protein is increased, and the sugar is diminished or absent. The meningococci may be seen in a direct smear mostly within the polymorphs, or they may be obtained on culture.

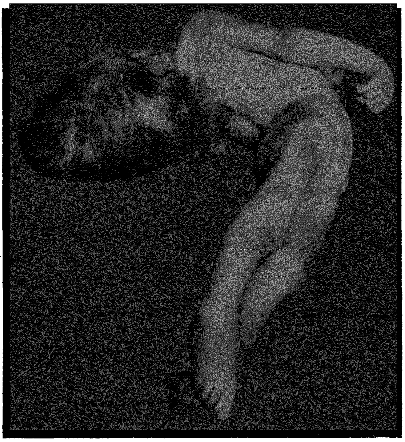

Fig. 80. An infant aged fifteen months suffering from meningitis, and showing severe head-retraction, rigidity of the limbs, and wasting.

Although stiffness of the neck must always raise the question of meningitis, it may be met with under other circumstances. In young children the onset of acute infections, such as tonsillitis, otitis media, or apical pneumonia, may be accompanied by evidence of meningeal irritation such as stiffness of the neck and even a positive Kernig's sign—a state of affairs termed meningismus—and the differentiation from meningitis may at times present great difficulty. If after a careful examination a doubt persists, the question can be settled by a lumbar puncture, for in meningismus the cerebro-spinal fluid, beyond being under slightly increased pressure, is normal. Head-retraction is also an occasional feature of mental defect in infancy, but the lack of acute illness and the history of a retarded mental development will prevent error.

Course. Meningococcal meningitis may at times be fulminating, killing within twenty-four hours, and is then apt to be accompanied by a rapidly developing purpura, with hæmorrhages also into the suprarenals, but often the illness drags on for from three weeks to three months. The acute stage subsides in about ten days, and in successfully treated cases recovery may then be looked for, although one or more relapses occasionally take place. In cases that last for two or three months, severe retraction of the head persists, vomiting becomes intractable, and gradually the child sinks into a state of progressive emaciation which may reach a truly remarkable degree. During this period the temperature is mostly subnormal, but may flare up for a few days at a time. Such cases usually terminate fatally, and the appearance of purpura is often a warning that the end is near.

Recovery does not necessarily mean a return to normal health, for unfortunately the illness may be followed by various sequelæ, of which the most important are hydrocephalus, spastic paralysis, blindness or deafness, and mental defect. The last shows all grades from imbecility to attacks of screaming and unmanageableness.

Treatment. The child should be nursed in a darkened room, and be protected from noise. Careful attention must be given to pressure points, and the use of a water-bed is helpful in preventing bed-sores. The diet must be fluid, and will consist mostly of milk and milk foods. If the illness becomes chronic, it may be necessary to feed through an œsophageal tube.

As in suppurative meningitis, chemotherapy with one of the sulphanilamides plays the predominant part in treatment. Sulphapyridine, sulphathiazole, and sulphadiazine seem on the whole to be equally effective, in fact results depend far more on maintaining an adequate dose over several days than on the particular preparation employed. As to dosage, the instructions on p. 715 should be followed, and a full daily dose should be persisted with until the temperature has fallen and the symptoms are subsiding. With early diagnosis, this usually occurs in from two to five days. Thereafter the dose should be reduced, but the drug should not be stopped until the temperature has been normal for a week, and the cerebro-spinal fluid has been proved to be sterile. Almost always the drug can be given by mouth, but should the child be too comatose to swallow, one of the soluble preparations should be injected intramuscularly every eight hours, the total daily amount being the same as by the oral route. When giving the injections, proximity to nerve trunks should be avoided owing to the risk of setting up a neuritis, with permanent paralysis as a possible sequel. Recent figures indicate that chemotherapy has reduced the mortality of this disease to about 10 per cent.

Since chemotherapy has proved so successful, treatment with antimeningococcal serum has been to a great extent abandoned, for the results obtained when both methods are combined are no better than when chemotherapy is used alone. If serum treatment is to be used, initial injections of 10 c.c. should be given intravenously, intramuscularly and intrathecally, the intrathecal injection being repeated daily for three days, and then on alternate days until the cerebrospinal fluid is sterile.

It sometimes happens that after a few days fluid cannot be obtained from a lumbar puncture, and there may be a coincident rise of temperature and an increase of meningeal signs. This is usually due to the exudate at the base of the brain having blocked the exits from the fourth ventricle. A cisternal puncture through the foramen magnum into the cisterna magna should then be carried out, and if fluid is easily obtained serum should be given by this route. In infants a better method is to puncture the lateral ventricle by going through the lateral angle of the fontanelle, serum being given by this route.

TUBERCULOUS MENINGITIS

This is the most frequent variety of meningitis in childhood, and in common with thoracic and abdominal tuberculosis, has its greatest incidence in the first two years of life. This is illustrated by the following chart, which shows an analysis of 126 consecutive cases from the post-mortem records of The Hospital for Sick Children. Of the 27 cases that occurred during the first year of life, none was under four months of age, and only 6 were under six months. That the incidence of tuberculous meningitis should be so great in the early years is due to the tendency of tuberculosis at this age to become generalised instead of remaining as a discrete lesion, and this in its turn depends on the fact that infection is occurring in a fresh soil in which there has been no time for previous infection, and therefore no opportunity for immunity to develop.

Tuberculous meningitis is never a primary disease, for invariably if a careful search is made at post-mortem examination a focus of infection is to be found somewhere else in the body, although there may be no evidence of such a focus during life. In two-thirds of the cases the primary infection is in the lungs or the mediastinal glands, while most of the remainder are accounted for by infection of the bowel and mesenteric glands. Until recently tuberculous meningitis has been supposed to come about by direct infection of the meninges by bacilli carried in the blood from some focus elsewhere in the body, but recently Rich and McCordock[1] have produced strong evidence to show that the bacilli carried in this way produce first of all caseous foci in the brain, spinal cord, or adjacent bones, and it is only when these lesions rupture into the cerebro-spinal fluid that meningitis results.

Symptoms. The onset of tuberculous meningitis is insidious. Often the first thing to be noticed is a change in the child's temperament, making him irritable, listless, and refusing to play with his toys or his fellows. There may be sudden periods of drowsiness alternating with the child's normal alertness. Older children may complain of severe headache, and younger children may indicate their pain by sharp screams and by putting their hands to their head. Vomiting is as a rule an early symptom and generally occurs without relation to meal times, although this is not always so. Constipation is also usual, and together with headache and vomiting forms a triad of symptoms at all times suggestive of tuberculous meningitis. The child may remain in this state for a varying time up to three or four weeks, until he gradually passes into the next stage, in which evidence of meningeal irritation appears.

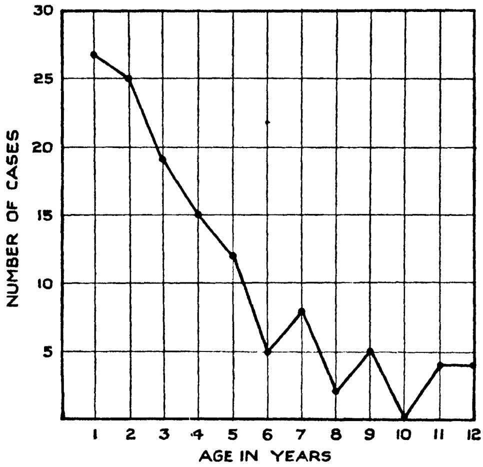

FIG. 81. Chart showing the age at death of 126 consecutive cases of tuberculous meningitis (see text).

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[1] *Bull. Johns Hopkins Hosp.*, 1933, Jan., p. 5.

In this stage—the stage of irritability—the child characteristically lies curled up on his side, disliking the light and strongly resenting examination, but otherwise lying drowsy or picking at his lips. At times the face may show sudden flushes, which last for a few minutes and then give way to pallor. The breathing may be irregular, and may be interrupted by frequent sighs. Evidence of an increased intracranial pressure is shown at this stage by a tense fontanelle and by a slowing of the pulse, which may drop to 60 beats a minute and is often irregular. Grinding of the teeth sometimes goes on for hours at a time. By this time examination generally shows some degree of stiffness of the muscles of the neck, so that the whole child may be raised by lifting the head, but the stiffness is seldom enough to cause any head-retraction. Kernig's sign may or may not be present, and its absence in no way militates against the diagnosis. The tendon reflexes are, as a rule, exaggerated, the plantar responses are variable, and a tâche cerebrale may be detected by gently stroking the skin of the abdomen with a finger-nail—within a minute or so the site of the stroke is marked by a broad pink flush, which persists for several minutes. A squint, usually an internal strabismus, is now likely to appear, and there may be some ptosis of the eyelids.

Although the above description suggests that there are numerous signs of the disease, actually clear evidence of meningitis is often difficult to establish, and the diagnosis at this stage depends more on the general appearance of the child and the accumulation of small clinical points than on any one well-marked symptom.

The stage of irritability may last up to a week before the child passes into the final paralytic stage. In this stage the child comes to lie on his back, with the hands folded across the abdomen and perhaps picking at the umbilicus. The irritability is replaced by coma, and the child may lie for hours with eyes staring vacantly and the pupils dilated. The weight rapidly goes down, and the abdomen becomes hollowed out and the skin loses its elasticity. The pulse now quickens to 120 or more per minute, and is irregular, and the respirations increase and may be accompanied by coarse rattles in the trachea. The tendon reflexes may be absent, there is incontinence of urine and fæces, and fine tremors of the limbs are frequent, or occasionally there may be choreic movements. Convulsions are very likely at this stage, and may become almost continuous and are accompanied by much sweating and a high temperature. Towards the end of the disease the vessels in the optic fundi become turgid and engorged, and there may be papilloedema. Tubercles develop on the choroid in a small proportion of cases, and when the diagnosis is in doubt the fundi should always be examined, since the presence of choroidal tubercles makes the diagnosis certain. They appear as yellowish-white circular patches, hardly as large as the disc, with a fluffy border, and situated as a rule by the side of a retinal vessel. Strictly speaking, they are evidence of a generalised miliary tuberculosis, of which the meningitis is but a part. During the final twenty-four hours sugar may appear in the urine, although by this time the diagnosis is so obvious that the glycosuria has no clinical importance. The paralytic stage lasts about four or five days and invariably ends in death.

The temperature chart follows no regular pattern. As a rule the temperature is normal or raised only one or two degrees until the last day or so, when it rises rapidly and the child may die in hyperpyrexia. In other cases a temperature as high as 103° F. may be sustained throughout the illness.

Almost always the course is steadily downhill from the onset, but occasionally, even when the stage of coma has been reached, the child may recover consciousness and for a day or two there may be a spell of lucidity and the child may sit up and play with his toys, but inevitably coma returns and death ensues.

Diagnosis. The diagnosis presents the greatest difficulty in the early stages, at a time when the symptoms are vague and amount to little more than irritability, listlessness, constipation, and perhaps headache, and when physical signs are indefinite. It is to be remembered that the classical signs of meningitis may not appear until the last few days of the disease, indeed the presence of tuberculous meningitis will often be suspected in its early stages on the history and general attitude of the child, coupled with the lack of pronounced meningeal signs.

Even in the early stages the cerebro-spinal fluid may show sufficient change to enable the diagnosis to be made with tolerable certainty, and therefore a lumbar puncture should be performed. The fluid is under increased pressure, and is generally clear and colourless. If allowed to stand for a few hours a fine cobweb-like clot forms from which tubercle bacilli can often be recovered if a careful search is made. The cells are increased to 30 to 200 per c.mm. and are mostly lymphocytes. Occasionally the course of the illness is more rapid and lasts only ten days or so, and in such cases the fluid may be opalescent and the cells chiefly polymorphs. The protein is raised, the sugar is normal or diminished, but the most important finding consists of the lowered chloride content, which from the very beginning drops from the normal figure of 0·75 per cent., and continues to fall until it may be below 0·6 per cent. A chloride value between 0·7 and 0·65 per cent. is very suggestive of tuberculous meningitis, and below this level is absolutely diagnostic.

Various conditions may be confused with the early stages of tuberculous meningitis. In infants, such causes of cephalic irritation as teething and otitis media are possible pitfalls ; apical pneumonia, acute pyelitis, and acute uræmia may also lead to confusion, but should be excluded by a thorough examination of the lungs and urine. An attack of cyclic vomiting must also be differentiated by examination of the urine for acetone and by the history of similar attacks having occurred before. The various forms of encephalitis in childhood may also give rise to difficulty, but the more rapid onset and the cerebro-spinal fluid findings will serve to distinguish these from tuberculous meningitis.

Morbid Anatomy. At post-mortem examination the brain appears swollen and the convolutions may be slightly flattened. On the base of the brain over the middle and posterior thirds there is a thin yellow gelatinous exudate, spreading laterally into the fissures of Sylvius and extending posteriorly between the pons and the undersurface of the cerebellum. Miliary tubercles can generally be made out with the naked eye, and are seen to best advantage alongside the vessels in the fissures of Sylvius or around the basilar artery. On opening the brain the ventricles are found distended, and one or more tuberculous caseous masses may be found embedded in the cerebral substance.

Almost always an old tuberculous focus is present in some other part of the body, usually in the mediastinum, and in the majority of cases a few miliary tubercles can be seen in other organs, such as the liver, spleen, and kidneys.

Treatment. Tuberculous meningitis is invariably fatal, and treatment can only be directed to the relief of symptoms. In the early stages headache may be relieved by phenazone (gr. 3 to 5) or phenacetin (gr. 4) and caffeine (gr. 1). Points to which special nursing attention must be paid are the emptying of the bladder, and the care of the eyes when the child is in coma-vigil. Convulsions often arise towards the end of the disease and are most distressing to the relatives ; if sedatives do not control them they can be kept in check by the withdrawal of cerebro-spinal fluid.

Acute Aseptic Meningitis (Serous Meningitis)

This is a benign form of meningitis, which was first clearly described by Wallgren in 1924, and which may occur sporadically or in small epidemics. Its characters comprise an acute onset with fever, and the rapid development of stiffness of the neck, a positive Kernig's sign, photophobia, and sometimes a squint and convulsions. The cerebro-spinal fluid is clear, and shows an increase of cells, mostly lymphocytes, usually to about 200 but occasionally up to 2,000 or 3,000 per c.mm.; the chlorides and sugar are normal and the fluid is sterile. The condition may easily be mistaken for tuberculous meningitis, but the normal chloride content of the spinal fluid is an important distinguishing feature. The pre-paralytic stage of acute poliomyelitis may also present a closely similar picture.

Following the experiments of Armstrong and Lillie,[1] the disease is held to be caused by a filtrable virus, which is harboured by mice and is capable of producing a lymphocytic meningitis in mice and monkeys; these animals can however be protected by the serum of human convalescents.

The prognosis is favourable. The meningeal symptoms subside on the withdrawal of cerebro-spinal fluid, the lumbar puncture being repeated once or twice if necessary.

HYDROCEPHALUS

Hydrocephalus is a condition in which there is an excessive accumulation of cerebro-spinal fluid inside the skull. This may come about either from over-production of fluid or from failure of the fluid to be absorbed. In order to understand how hydrocephalus may develop, the normal circulation of the cerebro-spinal fluid must first be described. The fluid is secreted by the choroid plexuses chiefly into the lateral ventricles, whence it flows *viâ* the foramina of Monro into the third ventricle, and from there through the iter into the fourth ventricle. It then escapes from the ventricular system through the foramina of Magendie and Luschka into the subarachnoid space at the cisterna magna. A small amount flows down the spinal subarachnoid space, but the majority passes upwards over the brain, reaching the subarachnoid space over the hemispheres by passing round the free edge of the tentorium cerebelli. Absorption of the cerebro-spinal fluid takes place chiefly by way of numerous arachnoid villi, which project from the dura into the main cerebral sinuses.

Hydrocephalus in childhood is almost always due to deficient absorption of the cerebro-spinal fluid, and this may come about either because the fluid cannot escape from the ventricular system (*obstructive or non-communicating type*), or because of disease of the absorbing surface in the meninges, usually inflammatory. This latter variety is spoken of as the *communicating type*.

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[1] Armstrong, C. and Lillie, R. D., *U.S. Pub. Health Rep.*, 1934, **49**, 1019.

The investigation of the circulation of the cerebro-spinal fluid may be carried out by withdrawing 2 c.c. of fluid through a ventricular puncture and injecting the same amount of a neutral solution of indigo-carmine or phenol-sulphone-phthalein. Patency of the foramina of Monro can be proved by obtaining the dye from a ventricular puncture on the opposite side, while the presence of the dye in the fluid obtained from a lumbar puncture shows that the cerebro-spinal fluid is circulating out of the ventricular system. As a rule, in the obstructive type of hydrocephalus only a small amount of fluid can be obtained by lumbar puncture and it is not under increased pressure, while in the communicating variety the fluid is under pressure, and if much is withdrawn the fontanelle will collapse. The position of the obstruction can sometimes be localised by ventriculography, which consists of X-raying the head after withdrawing cerebro-spinal fluid from the ventricles and replacing it by air or oxygen.

Hydrocephalus may be a congenital condition or may be acquired after birth. Of the causes which arise at or before birth, deformities in the ventricular system such as obliteration of a foramen of Monro or of the iter account for some, while others may be due to an intra-uterine meningitis. Another group of congenital cases is associated with spina bifida. Intracranial hæmorrhage produced during parturition and situated at the base of the brain may give rise to cicatricial tissue in the neighbourhood of the fourth ventricle and so cause an obstructive hydrocephalus.

Of the causes that arise after birth, meningitis is by far the most common. Tuberculous meningitis is almost invariably accompanied by dilatation of the cerebral ventricles as a result of blocking of the openings from the fourth ventricle by exudate, and the same process often occurs in meningococcal meningitis, particularly in the posterior basic form in infancy, in which condition hydrocephalus is a well-known sequela. Exudate may also produce matting between the free edge of the tentorium and the brain stem and so prevent the cerebro-spinal fluid from reaching the absorption area over the hemispheres, or if the exudate is spread over the hemispheres it may lead to interference with the function of the arachnoid villi, from which the cerebro-spinal fluid is normally absorbed into the cerebral sinuses.

Two other causes of hydrocephalus are syphilitic meningitis, which, at any rate in infancy, usually occurs at the base of the brain ; and tumours, when so situated as to interfere with the circulation of the cerebro-spinal fluid.

At post-mortem examination the brain may be found enormously distended. The gyri are flattened and the sulci obliterated. The cerebral tissue becomes very thinned out, and in severe instances may only measure a few millimetres in thickness. In infancy the skull bones may become so thinned that during life they can be indented by pressure of the finger, giving the physical sign of craniotabes.

Clinical Appearance. Hydrocephalus occasionally arises before birth, and may then give rise to so much difficulty during parturition as to call for craniotomy. During infancy, when the fontanelle is still open and before the sutures of the skull are firmly united, the increased intracranial pressure forces apart the bones of the vault so that the head becomes obviously enlarged. Although the fontanelle normally closes at about eighteen months, the union of the sutures is not yet firm, and under conditions of increased intracranial tension the sutures and fontanelle may re-open until the third or even fourth year.

The enlarged head of hydrocephalus is globular in shape, the forehead bulges forwards and the parietal bones bulge laterally above the ears. The area of the anterior fontanelle becomes much enlarged, stretching almost from ear to ear and extending backwards to the open posterior fontanelle. As a rule dilated veins can be seen coursing down the sides and front of the scalp. The huge head is in striking contrast to the small face and usually wasted body of the infant. Pressure on the orbital plates causes the eyes to be down-turned, sometimes to such an extent that the child has to learn to pull down the lower eyelid in order to see. Blindness may occur from stretching of the optic nerve, causing a primary optic atrophy, nystagmus

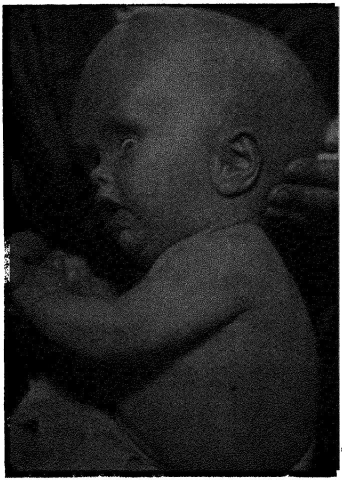

FIG. 82. Hydrocephalus in an infant aged 2½ months. Note the down-turning of the eyes.

is often present, and spastic paralysis of the limbs with increased tendon reflexes occurs in the more severe cases, and is associated with mental defect. In progressive cases the infant lies apathetic, and there is likely to be intractable vomiting.

Course and Prognosis. The majority of cases of hydrocephalus are progressive, and death takes place after an illness of weeks or months. The march of events is sometimes irregular, and measurement of the head at weekly intervals may show periods in which it ceases to enlarge. Occasionally the distension undergoes a permanent arrest, as though a satisfactory balance had been struck between the secretion and absorption of cerebrospinal fluid, and then the ultimate prognosis will depend upon the amount of cerebral degeneration which has taken place. Even when arrest occurs early, there is generally some degree of mental impairment, and the infant is likely to grow up into a simple-minded child, easy to control and of a pleasant disposition, but often handicapped by some degree of spasticity. There is always difficulty in controlling the large and heavy head, and because of this even the mildest cases are late in learning to sit up and walk. Should arrest occur at a later stage, all degrees of backwardness up to complete idiocy may follow

Regular measurements of the maximum circumference of the head enable one to tell whether the hydrocephalus is becoming arrested, and offer the best guide in attempting to form a prognosis. Normally at birth the maximum circumference is 13 inches, and this increases to 18 inches at one year old and 19 inches at three years.

Diagnosis. As a rule the striking appearance of a hydrocephalic child makes the diagnosis obvious, and difficulty is only likely to be met with in the mildest cases. In rickets the head often appears large, and a slight increase in the measurement above the normal may actually exist, but the shape is more square than globular and the top of the head is flattened. The head may also appear large in achondroplasia by comparison with the short limbs, and again measurement may show a slight degree of actual enlargement, but the characteristic dwarfing of the limbs will prevent error.

Treatment. Medical treatment is of little use except in cases due to syphilis, which often respond well to anti-syphilitic treatment. When the cause of the condition is in doubt, the Wassermann reaction should be done.

Various surgical manœuvres have been attempted, with only occasional success. Before deciding upon the particular operation, the type of hydrocephalus—whether communicating or obstructive—should be determined. In the communicating variety, when the enlargement of the head is only increasing very slightly, repeated lumbar punctures may be of service. In an attempt to diminish the production of cerebro-spinal fluid ligature of the carotid arteries in the neck has been performed, occasionally with beneficial results. With the same object, the choroid plexus has been removed, but it is a much more severe operation. In the obstructive type attempts have been made to create an artificial communication between the ventricles and the subarachnoid space by such methods as puncturing the corpus callosum, or the insertion of cannulæ, but the results are generally disappointing.

Acute Toxic Hydrocephalus. Instances of acute hydrocephalus are occasionally met with, mostly during childhood, in association with acute infection in neighbouring situations such as the pharynx, nasal sinuses, and particularly the middle ear (otitic hydrocephalus). The onset is rapid, and is indicated by headache, vomiting, and progressive drowsiness. Papillœdema is always present and may reach a severe degree, but other nervous manifestations are infrequent, and the customary signs of meningitis are wanting. The cerebro-spinal fluid is under considerable pressure but is otherwise normal.

Any focus of infection must receive immediate treatment. The withdrawal of cerebro-spinal fluid by lumbar puncture should be repeated daily or on alternate days until the pressure is no longer raised. The outlook as regards life is favourable, but the papillœdema may take some weeks to subside, vision remaining imperfect.

INTRACRANIAL TUMOURS

Intracranial tumours may occur at any age, being met with even in infancy, but they are rare under three years and only about a fifth of the total cases occur in childhood. In contrast to adults, the majority of tumours in children are subtentorial instead of supratentorial, and the most common situation is the cerebellum, especially the vermis cerebelli. Of the supratentorial tumours, those that arise in connection with the craniopharyngeal pouch form a special group (for pituitary adamantinomata and cysts, see p. 502). In the hemispheres the frontal and fronto-parietal lobes are most commonly involved. With regard to the type of tumour roughly one-third consist of caseous tuberculous masses—tuberculomata—but these are often so small as to give no indication during life of their presence. Of the tumours that regularly produce physical signs, gliomata are by far the most common. Tumours of the acoustic nerve, of the pineal gland, and angiomata are all rare forms.

The symptoms and signs are much the same as in adults, and therefore will not be detailed here. The classical triad of symptoms denoting an intracranial tumour, namely, headache, vomiting, and papilloedema, occur in older children in the same proportion of tumours as in adults, but in infants and young children up to about four years of age the first effect of a rising intracranial pressure is to distend the fontanelle and re-open the cranial sutures, and therefore at this age an expansion of the skull may take the place of headache, while vomiting and papilloedema may also be delayed. When vomiting occurs it usually does so quite suddenly without previous retching, and may show no relation to meal times. It is generally an early and frequent symptom of subtentorial tumours. Papilloedema is very variable; it usually appears early in cerebellar tumours, but is often very late in pontine cases.

In addition to the above, the signs of a cerebellar tumour include ataxy of the limbs, as shown by intention tremor and a reeling or staggering gait, loss of muscular tone and diminution or loss of the tendon reflexes, and nystagmus. An early weakness of one or both sixth nerves is often present, giving rise to an internal strabismus. It is due to stretching of the nerve and is not of much localising value. If the tumour occupies one-half of the cerebellum the signs are more marked on that side of the body, the nystagmus is slower towards the side of the lesion, and the child tends to fall towards that side. With a tumour placed in the vermis the signs are disposed more equally on the two sides, nystagmus may be absent, and the head may be held slightly retracted.

Pontine and medullary tumours cause paralysis of the cranial nerves in the particular region involved, and thus may affect the fifth to the twelfth nerves, and in addition the pyramidal tract is affected, giving rise to a contralateral spastic hemiplegia with increased tendon reflexes and an extensor plantar response. Should the tumour spread into both sides of the brain stem a quadriplegia will result.

Tumours in the frontal lobe give rise to spastic paralysis from involvement of the Rolandic area, the face, arm or leg being first affected according to the area of cortex concerned, and fits are also likely and may be either generalised or Jacksonian in type. With a left-sided tumour, provided the child has learned to talk and is right handed, aphasia may occur. Should the parietal lobe be affected, an opposite hemianæsthesia is produced. A combination of hemiplegia, hemianæsthesia and hemianopia points to the tumour being situated in the region of the internal capsule. Ocular paralysis (excepting sixth nerve paralysis) and rhythmical tremors indicate a deep lesion in the neighbourhood of the third ventricle, iter, or red nucleus.

Pituitary Adamantinomata

These are often spoken of as congenital tumours, although they grow so slowly that symptoms seldom arise until several years have elapsed. Even when symptoms have appeared they may remain stationary for years, and local pressure effects on the optic tracts are usually delayed until adolescence or adult life. The situation of the tumour being just above the pituitary body, symptoms arise partly from pressure on the pituitary and optic tracts and partly from interference with thalamic function.

The earliest symptoms are obesity with an increased sugar tolerance, attacks of drowsiness, and severe headaches. The epiphyses tend to unite early, so that some stunting of stature gradually becomes apparent, and the sexual changes at puberty are delayed. There may be attacks of uncontrollable thirst and polyuria. Interference with vision may be an early or a late sign ; the most common effect is bitemporal hemianopia, but squints and diplopia may occur. Examination of the fundi may show either papillœdema followed by secondary optic atrophy or a primary optic atrophy. The tumour tends to undergo cystic degeneration, and calcium salts may be deposited in the cyst wall, and when this happens an X-ray of the skull shows opaque flecks above the sella turcica. The sella itself becomes flattened out and the clinoid processes may appear eroded. These changes are usually deferred until young adult life.

Treatment. In the majority of cases surgical treatment offers the only hope. It is quite exceptional for a gumma to give the signs of a cerebral tumour during childhood, but a Wassermann reaction should be done, since a positive result would indicate antisyphilitic treatment. Otherwise operation should be performed in all cases in which there is any hope of the tumour being reached. When sight is beginning to fail a simple decompression may not only preserve the sight, but may relieve much of the headache.

Occasionally operation discloses a cyst which it may be possible either to remove or tap.

Tuberculomata

These are more common in childhood than at any other age. They appear as yellow caseous masses embedded in the substance of the brain or less commonly in the spinal cord, and they may sometimes show areas of healing by calcification. They are often multiple, and vary from the size of a pea to a walnut. They are most frequently situated in the cerebellum and brain stem, and probably because of their small size they seldom give rise to localising symptoms, but are either discovered unexpectedly at post-mortem examination, or, by leaking into the meninges, give rise to tuberculous meningitis. It is exceptional for the diagnosis to be made during life, but the localising signs of a tumour, together with evidence of active tuberculosis elsewhere in the body, would be very suggestive. In such cases treatment should be along the general lines for tuberculosis, including absolute rest, fresh air, and a nourishing diet. Operation should be avoided, owing to the risk of precipitating tuberculous meningitis.

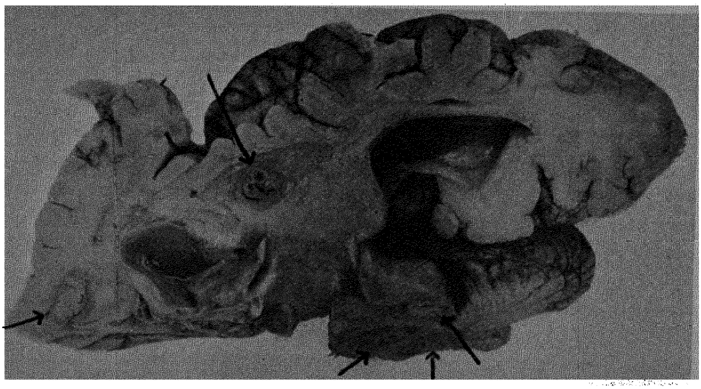

FIG. 83. Brain from a child aged six months, showing tuberculomata (see arrows) in the hemispheres and brain stem. The latter had obstructed the flow of cerebro-spinal fluid and had thereby caused distension of the ventricles.

Intracranial Abscess

Intracranial abscess is usually a sequel to ear disease, the abscess being situated either in the temporo-sphenoidal lobe or in the lateral lobe of the cerebellum on the same side as the affected ear. An abscess in the former position gives rise to signs of pyramidal involvement, while in the latter situation the signs are the same as those of a cerebellar tumour. When papillœdema is present it may be more marked on the side of the abscess.

Cerebral abscess may sometimes arise secondarily to bronchiectasis, or may be associated with pyæmia. The localising signs are the same as those of a tumour, but the onset is likely to be more acute and the march of events more rapid. Occasionally chronic encapsulated abscesses occur and are indistinguishable from tumours until the time of operation.

The treatment is surgical.

Spinal Tumours

These are rare in childhood. A meningeal sarcoma is said to be the most frequent variety. The signs do not differ from those that obtain in adults. Care must be taken not to mistake for a spinal tumour the more common compression paraplegia due to tuberculous disease of the spine.

CONVULSIONS

Convulsions may occur at any age, but are most common in the early years at a time when the nervous system is relatively unstable, and unduly responsive to small stimuli. It must be remembered that convulsions do not constitute a disease, but merely rank as symptoms ; in childhood they arise from numerous causes, and although they are epileptiform in character they are seldom due to idiopathic epilepsy. The term epilepsy indicates a condition in which fits, arising from no ascertainable cause, recur at varying intervals over a long period, and it follows that the occurrence of a single fit or even of a rapid burst of fits over a short period does not amount to epilepsy. The majority of fits occurring during the first two or three years arise from some cause or other which careful investigation will reveal. Fits of this nature will be dealt with before considering idiopathic epilepsy.

The characters of a convulsion are well known. The onset is sudden, the child stiffening, perhaps falling, becoming cyanosed and unconscious. The eyes roll up under the upper lids, the hands are clenched, with thumbs turned into the palms, and the legs are extended. After a few seconds the eyes and limbs begin to twitch, and then gradually the movements pass off and the child either returns to consciousness or may sink into sleep.

The sphincters are often relaxed during an attack and the tongue may be bitten. A fit may last from a few seconds up to half an hour and may be solitary or may be repeated several times, and in the most severe cases fits may succeed each other so rapidly that the child passes into a condition of status epilepticus.

Fits occurring within a few hours or days of birth are generally associated with a difficult or precipitate labour and are due to cerebral œdema or hæmorrhage. The fits are likely to recur for a few days, or when the injury has been more severe they may continue at intervals for months or years and may be associated with spastic paralysis and mental defect.

The association of fits with rickets has already been mentioned when dealing with that disease. The fits are then a manifestation of spasmophilia, and may be associated with laryngismus stridulus and tetany, and depend upon a lowered blood calcium. Fits arising between four and eighteen months of age should always prompt a careful examination for rickets and for latent tetany.

In young children, especially those of a sensitive nervous temperament or born of nervous stock, fits are easily provoked by some focus of irritation outside the nervous system. Fits of this nature may be termed " reflex " and may arise from many causes. In infancy constipation is sometimes the underlying factor, and it is not unknown for a burst of convulsions to cease with the passage of a big hard stool. At this age also the onset of feverish conditions such as pneumonia, pyelitis, gastro-enteritis, or otitis media, is likely to be heralded by one or more fits. Convulsions of this nature are seldom serious, certainly they are of less import than are fits arising later in the illness, for then they may indicate the development of some organic nervous complication. In highly strung infants even the worry of teething may be enough to provoke a fit, although it should never be assumed that teething is the cause of a convulsion until a most careful search has excluded other possibilities. Uræmic convulsions may be met with at any period in childhood, but are rare in infancy.

Convulsions may also be symptomatic of some intracranial lesion, and then may be termed " symptomatic " in contrast to " reflex " convulsions. Thus the onset of posterior basic meningitis is commonly accompanied by several fits, and numerous convulsions may occur in the later stages of tuberculous meningitis, while cerebral tumours, and the various forms of encephalitis, or intracranial vascular lesions, may give rise to symptomatic fits. Under conditions of severe cerebral congestion fits may occur such as those already mentioned in connection with asphyxia in the newborn ; in this group would also be placed fits which arise during severe bouts of whooping cough, or in the cyanotic attacks which may complicate congenital morbus cordis.

Convulsions in childhood may also be associated with mental deficiency, especially that form which is due to injury to the brain during birth, although mentally defective children of all types and grades are more prone to convulsions than are normal children. The question will often arise whether the mental deficiency is due to the fits, and it is often a most difficult question to answer, but as a rule both the fits and the mental defect have a common basis, and generally it may be taken that the mental deficiency was present beforehand and was only brought into prominence by the fit. Repeated convulsions over long periods, such as occur in idiopathic epilepsy, may be followed by progressive mental degeneration.

It seldom happens that death occurs during a convulsion. As a rule a fit is succeeded by a period of drowsiness, and the child may sometimes emerge from this with symptoms of paralysis of one or more limbs. The paralysis is generally temporary—Todd's paralysis—complete restoration of function occurring after a few days, but occasionally permanent damage is done.

Treatment. The usual method of checking a fit is to hold the child in a warm bath, or preferably a mustard bath, until the movements diminish, but obviously this can only be done when the fits last long enough for a bath to be prepared. Should the convulsions persist, they can be checked by a light chloroform anæsthesia, or by lumbar puncture and the withdrawal of cerebrospinal fluid. If the child is able to swallow, sedative drugs should be given, and chloral hydrate is the most valuable. Under three months of age gr. ½ to 1 should be given every two hours, until the fits cease ; from three to twelve months the dose should be gr. 2. If the infant is unable to swallow, double the dose of chloral hydrate should be given per rectum and may be combined with an equal amount of bromide. When the convulsions have ceased the dose of chloral hydrate should be reduced to gr. ½ to 1 ter die, and this should be continued for a week or ten days. Meanwhile, in both reflex and symptomatic fits the underlying cause must receive attention.

Idiopathic Convulsions of Infancy

This heading comprises the group of fits described by the late

Dr. John Thomson. The fits begin during the first three or four months of life, and increase rapidly in number until as many as twenty to fifty occur each day. Unless energetic treatment is instituted the infant is likely to pass into a fatal status epilepticus. Treatment consists of putting the baby rapidly under the influence of chloral hydrate, the dose being steadily pushed until the fits cease. The doses may then be reduced, but must not be entirely discontinued for two or three weeks, otherwise the convulsions are likely to return. When once the convulsions have been thoroughly controlled they are not likely to return in later childhood.

The possibility that fits of this type may be associated with tetany must be borne in mind, and corroborative evidence of tetany, or of early rickets, such as craniotabes or slight beading of the ribs, should be looked for. If there is reason to suspect tetany, an intramuscular injection of 10 c.c. of a 10 per cent. solution of calcium gluconate should be given in addition to sedatives by mouth.

Crying Convulsions (Breath-holding Attacks)

Fits of this nature are mentioned because they are so likely to be mistaken for epilepsy, but a careful history will soon show their distinguishing features. Crying convulsions generally first appear between nine months and two years of age, and the history is that the child has a sharp bout of crying, often associated with a display of temper. The crying lasts for a minute or two, and then the child holds his breath until he becomes severely cyanosed, and eventually passes into an asphyxial convulsion. In the fit he takes a breath, which relieves his asphyxia and so terminates the attack. Commonly the patient is an only child or has been spoiled, but this is not always the case. Unless the nature of the attacks is recognised they become more frequent until the child may have several during the day. Treatment with sedative drugs not only fails to control these fits, but by making the child depressed and miserable is actually likely to increase the number of attacks. If a careful history is taken there should be no difficulty in recognising fits of this nature, for the onset with severe crying and the story of breath-holding clearly demarcates them from ordinary convulsions.

The only way to thwart the fits is to make the child breathe as soon as he begins to hold his breath, and this is best done by sponging him unexpectedly with cold water or by a sharp slap, for if he can be made to breathe the cyanotic attack comes to an end. The purpose of the treatment must be thoroughly explained to the parents, since it is they who must deal effectively with the attacks. If treatment is properly and persistently carried out the attacks generally cease entirely within a few weeks.

Idiopathic Epilepsy

Epilepsy may occur at any age, and it has been estimated that about 1 case in 8 begins before the third year, but even so it is rarely possible to make the diagnosis at such an early age for the reason that one cannot be sure that the fits are going to recur and thus constitute epilepsy. On the other hand, it occasionally happens that fits which seem in infancy to have some reflex explanation continue in later life as idiopathic epilepsy. Even in older children epilepsy should not be diagnosed until a thorough search has excluded any other cause, since the diagnosis inevitably confers upon the patient a stigma of inferiority.

The seizures in epilepsy may consist of major attacks—grand mal—or minor attacks—petit mal. The appearance of a grand attack has already been described in the previous section. In about half the cases the fit is preceded by an aura, which may take various forms, either psychical, sensory, visual, auditory, or gustatory, and after the attack is over the child may fall into heavy sleep or may pass into a state of post-epileptic automatism, in which he may commit mischievous, spiteful, or cruel acts without being aware of or responsible for his actions. A feature of some cases of epilepsy is the tendency for the attacks to recur at the same time in the twenty-four hours, and inquiry should be made on this point, since if drug treatment is to be successful it is important that the drugs should exert their maximum effect at the time when the fits are expected.

The number of attacks of grand mal varies very much. Several may occur daily over long periods or there may be an interval of months between each fit, or they may occur in bouts of a dozen or so and then several months may pass before another bout occurs.

Petit mal is not uncommon in childhood, but is more easily overlooked than grand mal. Each attack lasts only a few seconds, the child stares vacantly, stopping whatever he is doing and seeming to lose himself, although he does not fall. Occasionally the eyes or limbs may jerk a little, and often the attack ends with a short sigh, and afterwards there may be a temporary pallor. The attacks are likely to be repeated frequently during the day, and two or three may be witnessed during the space of a consultation. When petit mal occurs in infancy, it is then often indicated by the infant, while sitting on his mother's knee, suddenly falling forward with his chin dropping on the chest and the hands being clenched. After a second or so he sits up and the attack is over. Parents are apt to think that the infant is playing, and do not realise the significance of these attacks.

Rarely, instances arise in which the ordinary petit mal is replaced by visceral manifestations. Thus one little girl observed by the author complained of sharp pains situated at the umbilicus and only lasting a few seconds. Examination failed to show any cause for these, and they gradually disappeared under treatment by luminal and bromides. Another boy six years old would suddenly stop and pass a little urine, no matter where he happened to be. These attacks also disappeared under treatment for epilepsy.

Status Epilepticus.

This condition is fortunately uncommon. In it the epileptic child has a burst of convulsions which recur so rapidly as to merge one into another, so that the child remains unconscious for hours or days, undergoing clonic movements. The temperature rises rapidly and is likely to become hyperpyrexial, and the attack may terminate fatally from cardiac failure. Treatment is an urgent affair, as the convulsions must be controlled with the shortest delay (*vide* treatment).

Diagnosis. The description of the seizures will generally enable the diagnosis of epilepsy to be made without difficulty. The fits must be distinguished from fainting attacks, which are probably not as common in childhood as parents would have us believe, but which occur with increasing frequency as puberty is approached. The pallor, limpness, and lack of clonic movements in fainting attacks are distinguishing features. Another common story which may lead to confusion with petit mal occurs in chronic indigestion, in which the child is said to have attacks of turning " deathly white." But the attacks last too long for petit mal, there is no hint of vacancy about them, and inquiry will show other evidence of a disordered state of the digestion. Lastly, the appearance of concentration, the reddening of the face and the movements of the legs which occur in infants who masturbate may lead to an error in diagnosis. There is, however, no loss of consciousness, and the annoyance of the baby when the movements are checked, together with the character of the movements, are distinguishing features.

Prognosis. Except in status epilepticus, the immediate outlook for a child in a convulsion is good. The remote prognosis is a more difficult matter to decide, but in the majority the fits can be checked to a great extent, and often entirely abolished. So long as there are no signs of mental degeneration an optimistic outlook is justified, provided that regular medical supervision can be given. Occasionally the attacks seem to terminate automatically, although this should never be anticipated in giving a prognosis. Hereditary predisposition does not affect the outlook (Osler). Petit mal is generally more resistant to treatment than grand mal, and so carries a more unfavourable prognosis. The possibility of mental degeneration is a most important point; as a general rule the greater the number of fits the greater is the likelihood of mental degeneration, and petit mal is in this respect more unfavourable than grand mal. When mental degeneration has once commenced, even if the fits are checked the mental state does not improve, but continues to become slowly worse. On the other hand, attacks of grand mal at long intervals may go with a high level of intelligence.

Treatment. Should an attack of grand mal be witnessed, a space should be cleared so that the child does not injure himself, and a gag such as a clothes-peg put between the teeth to save the tongue from being bitten. If the fit continues for more than a few minutes a light anæsthetic may be given. In status epilepticus anæsthesia is very useful in stopping the fits, or a lumbar puncture may serve a similar purpose. As soon as the convulsions have been temporarily checked, sedative drugs should be given per rectum. For a child of five years, chloral (gr. 10) and potassium bromide (gr. 30) would be a suitable dose and should be repeated in six hours, or if consciousness is regained half this dose should be given by mouth every four hours for a day or two until, so far as one can judge, the tendency to further convulsions has passed.

General Treatment of Epilepsy. Attention to ordinary hygiene is important. Many epileptics are habitually constipated, and so the diet should be simple and the bowels carefully controlled. Regular periods of rest each day should be arranged, and it is important to avoid mental excitement. The worry of competitive education is harmful, and the majority of epileptic children will be better at a special school where learning is not at a premium, although this does not apply to the child who has only occasional fits, since he is usually able to hold his own quite well at an ordinary school.

Dietetic Treatment. Until recently the dietetic treatment of epilepsy has received scant attention, but successful results have lately been recorded following the use of a ketogenic diet. The diet seems to be of greatest service in cases of petit mal, which is generally the type of epilepsy most resistant to drug treatment. Talbot [1] and his colleagues have estimated that this treatment gives curative results in as many as 30 per cent., although no such optimistic claims have as yet been made in this country. The principles of a ketogenic diet are that the carbohydrates should be reduced to a minimum and the fats should be pushed to excess. Such foods as milk, butter, cheese, cream, eggs, fish and meat form the bulk of the diet, the efficiency of the diet being shown by the appearance of acetone bodies in the urine. There is no doubt that the treatment is sometimes of considerable value, and should be given a trial when other methods have failed. The diet must be strictly adhered to, and in the author's experience it is essential to have the child in hospital. As the ketosis develops the temperature may rise, and the child is likely to appear very toxic ; in a successful case the fits cease after the ketosis has been maintained for a week or so, but the diet should not be relaxed for a further week, otherwise the fits may return. McQuarrie [2] has pointed out that during ketosis the water balance is disturbed and the tissues become less retentive of water ; he claims to have had successful results with epileptic children by severely limiting their intake of water, cutting it down as low as 30 c.c. per kilo of body-weight.

Drug Treatment. The object should be to employ drugs in a dose sufficient to abolish the fits, and then to continue with that dose for at least six months after the last fit. A trial period without drugs should then be allowed.

The two most useful drugs are phenobarbitone (luminal) and the bromides. If phenobarbitone is prescribed, the dose for a child of three years should be gr. $\frac{1}{4}$ twice a day, or three times a day from then until five years. In older children a dose of gr. $\frac{1}{2}$ thrice daily should not be exceeded. In the author's experience phenobarbitone is more successful when combined with the bromides ; for a child of five years the following mixture, given three times a day, would be suitable :—

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[1] Talbot, F. B., Metcalf, K. M., and Moriarty, M. E., *Amer. Jour. Dis. Child.*, 1927, **33**, 218.

[2] Irvine McQuarrie, *Amer. Jour. Dis. Child.*, 1929, **38**, 451.

℞ [1]Phenobarbitoni solubilis gr. ¼.
Potassii bromidi gr. iii.
Sodii bromidi gr. iii.
Syrupi simplicis m. xxx.
Aquam chloroformi ad ʒii.

The addition of 5 m. of tinct. belladonna to this mixture is often beneficial, especially in cases of petit mal. Another drug which has sometimes seemed valuable is sodium borate (gr. 2 to 4 ter die).

When ordering mixtures in epilepsy, inquiry should be made whether the fits occur at any particular time of the day or night, the doses being so timed as to have their maximum effect when the fits are expected. Thus in one child who had fits generally at about 4 a.m. success followed the administration of drugs at 4 p.m., 8 p.m., and midnight. It is also noteworthy that epileptic children will tolerate sedative drugs without showing any drowsiness in doses which would make an ordinary child thoroughly sleepy. When bromides are being given over long periods a watch must be kept for acneiform eruptions ; the addition of a minim of liquor arsenicalis to a bromide mixture will sometimes help to prevent these rashes. In some children bromides give rise to irritability, peevishness, or loss of appetite, and in that case they should be used alternately with other sedative drugs, such as urethane (gr. 5) or chloretone (gr. 3) three times a day. Either of these drugs may be given to children over five years of age.

Variants of Epilepsy

Jacksonian fits are characterised by beginning in some local group of muscles, such as the hand or arm, to which they may be confined or may spread to involve the whole body. Although they are sometimes of localising value, as for instance when a cerebral tumour affects the motor cortex, too much importance must not be attached to this in childhood, since they may sometimes replace ordinary fits, and also the point of origin of the fit may vary in different attacks.

Pyknolepsy. This is a rare condition characterised by the rapid development of petit mal attacks, which may reach as many as 50 or 100 in the day. The fits first appear about the time of the second dentition, and although the attacks become so numerous, mental degeneration does not ensue. The prognosis is good, the attacks ceasing spontaneously after a year or two. Treatment along the usual lies for epilepsy is generally ineffective, although success may follow the use of a ketogenic diet.

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[1] Phenobarbitone is insoluble in water, and if given in a mixture should be prescribed as the soluble sodium salt. Ammonium salts should not be combined in the mixture, as they throw the phenobarbitone out of solution.

Narcolepsy. This rare disorder generally affects adults, although instances in childhood have been recorded. The disease is characterised by sudden and irresistible attacks of sleep, which generally come on at times of emotional stress or excitement. In a boy observed by the author the first attack occurred at a cinema just as the climax of the performance was approaching. The sleep may last for a few minutes or for some hours, but the patient can generally be roused. Somewhat similar attacks called *Catalepsy*, occurring under similar circumstances, are characterised by the patient falling limply to the ground and being unable to move, although fully conscious of what is going on around him. The attacks last longer than petit mal, while consciousness is not lost as in grand mal. Attacks of this nature are sometimes associated with cerebral tumours, and may follow encephalitis lethargica.

Epiloia (Tuberose Sclerosis of the Brain) [1]

This rare condition is characterised by progressive mental degeneration, epileptic fits, and a rash on the face (adenoma sebaceum). Sometimes the first symptom is the rash, which appears at about four or five years of age, beginning in the naso-labial folds as pale pink spots at the openings of the sebaceous glands, and later coalescing into small red papules. The rash gradually spreads over the face. The mental degeneration slowly progresses to idiocy, and is accompanied by frequent small and large fits. The course terminates fatally after a few years. At post-mortem examination small nodules of neuroglial tissue are found scattered throughout the cerebral hemispheres, with destruction of grey matter. In addition, small fibromata may occur in the viscera or skin.

Treatment is purely symptomatic. Owing to the severe mental degeneration, these cases generally find their way into institutions for the mentally defective.

INTRACRANIAL HÆMORRHAGE, THROMBOSIS, AND EMBOLISM

Hæmorrhage

Apart from hæmorrhage produced during birth, intracranial hæmorrhage is a rare event in childhood. In an analysis of 10,150 post-mortems at The Hospital for Sick Children[1] in children under twelve years of age, intracranial hæmorrhage was found in 50—less than 0·5 per cent. Although birth hæmorrhages were not included in this series, it was found that intracranial hæmorrhage was much more common in the first year than in later years. Just as birth hæmorrhages are situated more often in the meninges than within the brain substance, so, in contrast to adults, are intracranial hæmorrhages in childhood arising from other causes.

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[1] Brushfield, T., and Wyatt, W., *Brit. Jour. Dis. Child.*, 1926, **23**, 178-254.

Of the causes of intracranial hæmorrhage other than birth injury, trauma is one of the most common. Considering how frequently and often heavily children fall and hurt their heads it is perhaps surprising that traumatic hæmorrhage is not more often produced. As a rule the symptoms develop immediately after the accident, but occasionally there may be a latent period of a few days up to a few weeks between the time of the injury and the onset of symptoms.

One of the commonest causes of cerebral hæmorrhage in adults and one of the rarest in children is chronic interstitial nephritis. The various blood diseases, *e.g.*, purpura and leukæmia, occasionally give rise to hæmorrhage, which is usually meningeal, and cerebral hæmorrhage has also been recorded in infantile scurvy. The acute specific fevers, particularly whooping cough, have on rare occasions been complicated by meningeal or cerebral hæmorrhage.

Spontaneous subarachnoid hæmorrhage due to the rupture of a congenital aneurysm, situated usually on the Circle of Willis, has been recorded in children although it more commonly occurs in adults. The symptoms consist of severe headache and irritability or delirium followed in a few hours by coma and convulsions. Occasionally small hæmorrhages may be seen on the retinæ. A lumbar puncture shows the cerebro-spinal fluid evenly mixed with blood.

A fatal issue is likely, but a temporary recovery occasionally occurs, only to be followed in a few weeks or months by a fresh hæmorrhage. Lumbar puncture is a dangerous procedure in these cases, and as soon as the character of the fluid is seen the puncture should be stopped, otherwise fresh hæmorrhage may be caused.

The only case observed by the author occurred in a girl of thirteen years of age.

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[1] Sheldon, W., *Quart. Jour. Med.*, 1927, **20**, 353.

Pachymeningitis Hæmorrhagica Interna

This is a rare condition occurring generally in enfeebled infants. Rosenberg[1] in 1913 was able to collect 38 cases. At autopsy extensive blood clot is found on the inner layer of the dura mater. There may be several layers of clot in various stages of organisation and cyst formation, the whole being enclosed in a vascularised membranous sheath.

The symptoms are the outcome of successive hæmorrhages, and consist of convulsions followed by such signs of a raised intracranial pressure as bulging of the fontanelle and separation of the cranial sutures. Retinal hæmorrhages may be present together with some swelling of the discs, and the cerebro-spinal fluid may be discoloured by blood.

The cause of this remarkable condition is unknown. Recovery occasionally comes about, but the child is likely to be left with permanent paralysis. The treatment is symptomatic. Chloral should be given to control the convulsions, and excessive intracranial tension may be relieved by the cautious withdrawal of cerebro-spinal fluid.

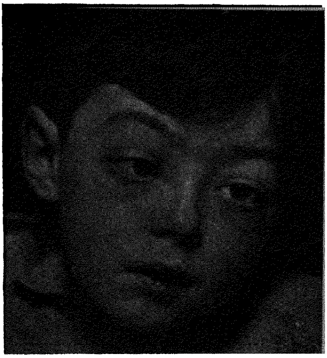

FIG. 84. Boy aged ten years, who died from cavernous sinus thrombosis. Note the proptosis and internal strabismus of the right eye.

Thrombosis

Intracranial thrombosis most commonly occurs in the lateral sinus, and is then secondary to ear disease. It is likely to cause some rigidity of the neck and there may be tenderness along the line of the internal jugular vein. Prompt surgical treatment is necessary. Thrombosis of the cavernous sinus is also occasionally met with, and may be the result of ear disease or of infections about the face. In addition to fever, vomiting, and rigors, there may be such localising signs as proptosis of one or both eyes, œdema of the conjunctiva, and ophthalmoplegia. These cases are almost always fatal.

Extensive thrombosis of the various sinuses and cerebral veins may sometimes be found at autopsy in infants who have died from severe wasting conditions such as chronic gastro-enteritis or after a prolonged attack of broncho-pneumonia. The formation of the thrombi is likely to be indicated during life by convulsions arising late in the course of the disease. Cerebral thrombosis may also arise in older children as a manifestation of congenital syphilis.

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[1] Rosenberg, O., *Berlin Klin. Wochenschr.*, 1913, **50**, **2272**.

Embolism

The most common cause of cerebral embolism is malignant endocarditis. The middle cerebral artery or one of its branches is usually affected, and extensive hæmorrhage into the substance of the brain is likely to result.

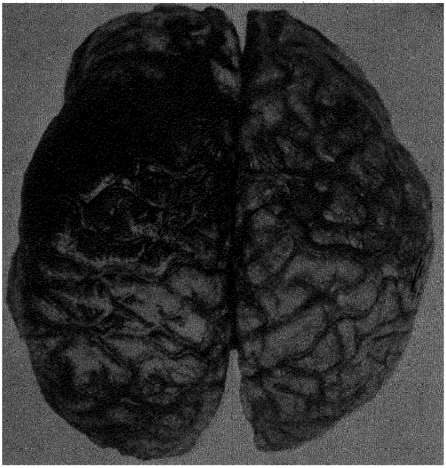

FIG. 85. Brain of an infant showing thrombosis of the cortical veins and hæmorrhage complicating broncho-pneumonia.

Symptoms. In many cases intracranial hæmorrhage or thrombosis is unsuspected during life, particularly when it occurs as a terminate event in the wasting diseases of infants. In young children convulsions are the chief manifestation; they have seldom a localising value, but in cases that survive long enough the paralysis which follows may indicate the site of the hæmorrhage. The onset of symptoms of cerebral irritation or of paralysis is sudden in cases of embolism, and generally rapid in hæmorrhage; when several hours elapse between the commencement of symptoms and the complete paralysis, thrombosis is more likely. If life continues for a few days the optic discs may show some swelling, and retraction of the head and Kernig's sign may develop. When the diagnosis of meningeal hæmorrhage is in question, help may be had from a lumbar puncture, for the fluid will be either diffusely blood-stained, or, if some days have elapsed, will show a yellow discoloration.

Treatment. This is largely symptomatic. Convulsions should be controlled with sedative drugs, but lumbar puncture as a method of treatment must be used with great caution. When the hæmorrhage is due to trauma and can be localised, the question of exploratory operation and removal of the clot must be considered.

CEREBRAL DIPLEGIA

Under this heading is included a heterogeneous group of cases manifesting a wide variety of paralytic symptoms, associated sometimes with abnormal movements of a choreiform or athetoid type, and usually accompanied by some degree of mental defect.

The causes of cerebral diplegia in young children may arise either before birth, during birth, or within a few months after birth. Our knowledge of pre-natal causes is scanty, but disease of the mother during pregnancy is probably the most important factor. Accidents and injuries to the mother's abdomen may sometimes damage the fœtal brain—this was so in an infant who died at birth and was found to have an old hæmorrhage in the right hemisphere which had resulted from a severe blow by a plank of wood on the mother's abdomen (Gibb, 1858). Old cerebral hæmorrhage has also been found in an infant delivered by Cæsarean section. A primary failure of cerebral formation—cerebral agenesis—is held to account for some, while fœtal cerebral thrombosis has also been recorded, leading to atrophy of the corresponding area of brain. Post-mortem examination in these cases is likely to show single or multiple areas where the cerebral tissue has been replaced by cystic spaces, which as a rule are not in communication with the ventricles—a condition to which the name "porencephaly" is given.

The majority of instances of cerebral diplegia in infancy arise during birth, and are due to hæmorrhage or bruising of the brain (*Little's Disease*). Injury by forceps and other manipulative measures account for only a proportion of these cases. An infant born prematurely is much more likely to suffer from cerebral trauma during birth than is one delivered at full term, probably owing to the increased fragility of the capillaries in the premature infant. Precipitate labour is always a dangerous event for the infant, while prolonged labour is also harmful, chiefly because of the severe cyanosis and cerebral congestion to which the infant may be subjected. That there is actual destruction of brain tissue was shown by McNutt,[1] who carried out post-mortem examinations on 34 cases of diplegia dating from birth, the patients having survived for various ages from fifteen months to seventy-three years. There was evidence in all of them of gross injury of the cerebral cortex, as evidenced by scarring, atrophy of

[1] McNutt, Sarah, *Amer. Jour. Med. Sci.*, 1885, **89**, 58.

the convolutions, depressions of the cerebral cortex, and cyst formation within the substance of the brain. The cortex may be thinned and there may be compensatory dilatation of the ventricles ; the circumference of the head is eventually likely to be below the average.

Infantile cerebral diplegia arising after birth may be due to a variety of causes, of which trauma, meningitis, vascular disturbances, and inflammatory conditions of the brain, particularly polioencephalitis, afford examples.

Symptoms. The most common form of cerebral paralysis in infancy is *spastic diplegia*, the outstanding feature being rigidity of the arms and legs. As a rule the legs are affected more than the arms and are held rigidly extended with the feet in a position of talipes equino-varus. Even in the mildest cases attempts to flex the joints will reveal the hypertonic state of the muscles. The spasticity of the legs is most easily shown by raising the child under the armpits ; the normal child draws its legs up, but in spastic diplegia the legs are stiffly extended and often crossed at the ankles owing to the spasm of the adductor muscles. Walking is always delayed, and is often characterised by a scissor gait in which the feet tend to cross each other. In more severe cases the child may be permanently bedridden. The legs are generally thin, but the circulation is unimpaired and the feet are warm, thus differing from the cold and bluish limbs in poliomyelitis. The tendon reflexes are generally exaggerated, but when the rigidity is extreme the reflexes may be difficult to obtain. The plantar responses are extensor.

The arms are affected to a less extent, and are held extended at the elbow, pronated, and with the thumb inturned across the palm. When the face is affected there is likely to be constant drooling of saliva and difficulty in swallowing. Strabismus is not uncommon, and blindness with

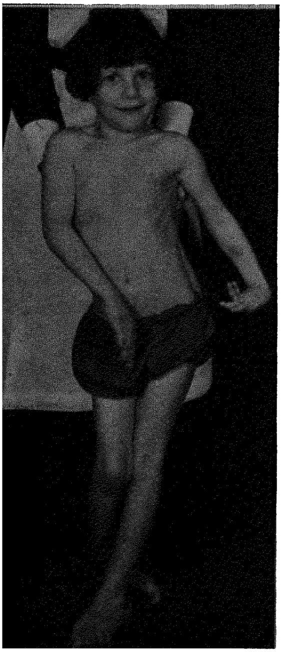

FIG. 86. Girl aged six years, suffering from spastic diplegia associated with athetoid movements.

optic atrophy, and deafness may also occur. While almost the whole child may in this way be affected, in other cases the spasticity may be confined to the legs (paraplegia) or perhaps to only one of the four limbs. Occasionally the distribution is hemiplegic.

Another clinical group is characterised by widespread muscular hypotonia. This may affect the limbs, making them flabby and enabling them to be placed in grotesque attitudes, but more usually the loss of tone affects the muscles of the back and neck ; the child is then late in learning to hold up its head and to sit up, while the gentle kyphosis of the normal infant's spine is exaggerated. The tendon reflexes in this group are generally absent. A third clinical group is marked by abnormal movements of the limbs. There may be slow twisting movements, particularly of the hands and arms (athetosis), or more rarely the movements may be choreiform. As a rule these movements are associated with increased muscular rigidity, increased tendon reflexes, and extensor plantar responses. The face may be affected, and the constant grimacing give an idiotic appearance, although children of this group sometimes show a surprisingly good level of intelligence.

The degree of mental deficiency depends upon the site and extent of the damage to the brain. The physical defect of spasticity is alone sufficient to make the child late in walking, but when the mentality is unimpaired the earnest efforts of the child to do as others is often quite striking. The majority however have some degree of mental deficiency, which varies from slight backwardness to idiocy. Unless the child with gross physical defect is well looked after, the intellect is sometimes needlessly dulled by reason of the lack of training on the part of those who have the care of the child. When the motor paths escape damage, mental deficiency may occur without accompanying physical impairment.

Convulsions occur in a high proportion of the cases. They may appear within a few hours or days of birth, or may be delayed for several months or even years, but the coincident evidence of organic nervous disease will at once separate them from cases of idiopathic epilepsy.

Diagnosis. The diagnosis of cerebral diplegia usually presents no difficulty. In mild cases the first thing to be noticed may be that the infant does not hold up his head or sit up at the usual time. In the group due to cerebral trauma at birth there may be difficulty in getting the newborn infant to suck, for some days crying may be absent or only feeble, and the fontanelle may be bulging and without pulsation. At that stage a lumbar puncture may confirm a diagnosis of hæmorrhage by showing the cerebral fluid to be discoloured with blood. Of the causes which operate after birth, congenital syphilis will be distinguished by other evidence of spirochætal infection and a positive Wassermann reaction, while in those instances that follow meningitis or polioencephalitis there may be a clear interval after birth in which the baby was well and moving its limbs normally until an acute illness supervened, probably accompanied by convulsions and retraction of the head, and followed by loss of use of the limbs.

Prognosis. As a rule paralyses and other physical defects are lasting, and although special methods of training may lead to some restoration of function, it is never fully regained. Athetoid and other bizarre movements generally persist, and the same is true also of the varying degrees of mental defect.

Treatment. Drugs are of very little value except in the control of fits, but in the absence of gross mental deficiency much may often be accomplished by assiduous training. During infancy full passive movements of the spastic limbs should be carried out regularly to prevent contractures, and later on active movements should be encouraged by a variety of devices. When the child has learnt to sit up he must be encouraged to walk round the furniture, and a heavy chair on wheels which he can push about is often a help. Co-ordination of leg movements can be encouraged by such exercises as making the child walk on different coloured squares marked out on the floor, or of the arms by simple games such as sorting out coloured balls and putting them in correspondingly coloured boxes. Much ingenuity can usefully be expended in working out such simple exercises appropriate to each individual case. For the group with atonic muscles massage is useful, but is contra-indicated in the spastic group. Orthopædic operations on the nerves and tendons are often of great service, particularly in helping the child to walk, but these measures should only be undertaken when the intelligence is sufficiently good for the child to benefit by them. As a rule they should be deferred until the child is three or four years old, by which time one may judge whether he is likely to be able to benefit by surgical procedures.

POLIOMYELITIS (Infantile Paralysis)

Poliomyelitis may be defined as an acute specific infection of the spinal cord and brain, characterised clinically by paralyses of sudden onset and varying distribution. The disease may occur sporadically or in epidemics.

Etiology. The disease is most common between the second and fifth years, but instances have been recorded within the first few months of life. The sex distribution is equal. There is a definite seasonal incidence, most cases occurring in the late summer between July and October.

The incubation period generally lies between two and seven days, but may extend to a fortnight. The virus is transmitted by droplet infection from the mouth or nose of infected persons, who may themselves not show symptoms, but act merely as carriers, although there is probably no doubt that a clinical case of poliomyelitis may also infect others at the very beginning of an attack before the paralytic symptoms have even appeared. It is probable that the virus soon dies out from the nasal secretions, so that the chronic carrier does not exist. That the infectiousness is short-lived is indicated by the fact that although children are often admitted to hospital wards within forty-eight hours of the onset of paralysis, a spread to other children does not occur.

Pathology. The virus discovered by Flexner and Noguchi in 1913, consisting of minute filter-passing globoid bodies, is now generally accepted as the cause of the disease. The infection gains access to the body through the nasal mucosa, and the nasal secretions of an infected case can be used to transmit the disease to monkeys. Whether the infection reaches the nervous system by a direct spread from the nose along the olfactory nerves or by an intermediate stage of general blood infection is not settled.

When death occurs during the acute stage of the disease post-mortem examination shows to the naked eye little in the nervous system beyond some slight congestion. Old-standing cases show some shrinkage of the cord in the affected region. Microscopic examination in the acute stage shows dilatation of the vessels of the grey matter, particularly in the neighbourhood of the anterior horn cells, and the capillaries are surrounded by a zone of small round cells. Within a few days the cells of the grey matter in the affected area undergo chromatolysis, and eventually become shrunken and absorbed. The peripheral nerves become atrophied, and the muscles supplied by them degenerate and waste.

Symptoms. The symptoms of poliomyelitis can most conveniently be grouped according to the area of the nervous system affected, but before doing so consideration must be given to the initial symptoms which may precede the appearance of paralysis. The recognition of this stage—the pre-paralytic stage—has recently been given prominence owing to the use of serotherapy, which to be effective has to be given in the incipient stage of the illness.

Pre-paralytic Stage. This stage lasts only a day or two, and is characterised by fever of three or four degrees, and often symptoms of nasopharyngeal catarrh resembling a common cold. Flushing of the face is often present at the onset, diarrhœa may sometimes be an early symptom, and there is likely to be headache and pain down the back. In other cases there may be vomiting and drowsiness or irritability. The most important sign, however, is stiffness of the neck, hardly amounting to actual head-retraction. In a sporadic case these symptoms are likely to be so vague as to be overlooked, but during the course of an epidemic, or during the summer months when the disease should be constantly in mind, if any doubt is entertained as to the possibility of poliomyelitis a lumbar puncture should be carried out, because there may be characteristic changes in the cerebro-spinal fluid even before the appearance of paralysis. The cerebro-spinal fluid is clear, under increased pressure, and the number of cells is increased to 50 to 200 per c.mm., the cells being either polymorphs or lymphocytes. The protein is slightly increased, but the chlorides are normal, which is an important point in distinguishing these cases from early tuberculous meningitis.

Spinal Form. This is by far the most common form of infantile paralysis. Within a day or two of the initial symptoms paralysis appears, and may come on with remarkable suddenness, perhaps during the daytime or perhaps when the child attempts to get out of bed in the morning. In sporadic cases the initial (pre-paralytic) symptoms may be absent, or else overlooked, and then paralysis will be the first symptom. Except in the rare spreading variety, the extent of the paralysis is greatest at the time of its first appearance, and the muscles of one or more limbs, or isolated groups of muscles, may be involved, together perhaps with the muscles of the trunk. The paralysis is from the first flaccid, with loss of tendon reflexes. Sometimes the affected limbs are acutely tender, but this passes off after a few days, and permanent sensory changes do not occur. The sphincters are not involved. Generally after a week or two recovery begins to appear, and the paralysis, which may at first have been widespread, becomes confined to perhaps one limb or even a single group of muscles. The legs are most commonly affected, and the muscles of the anterior tibial group are particularly vulnerable. The paralysis may show a symmetrical distribution. Wasting of the paralysed muscles becomes apparent within two or three weeks, and the circulation in the limb becomes impaired, as indicated by increasing coldness and cyanosis. Unless active treatment is instituted, the muscles normally opposed to the paralysed group will gradually cause contractures and deformities. The growth of the limb is likely to be interfered with, and some degree of shortening may eventually become apparent.

Occasionally the muscles of the abdominal wall are affected, causing considerable abdominal distension, or the intercostal or erector spinæ muscles may be involved, giving rise to respiratory embarrassment, or causing severe scoliosis. Paralysis of the cervical sympathetic, with narrowing of the palpebral fissure, small pupil, and flushing of the affected side of the face, is a rare form of the disease.

Meningeal Form. Occasionally meningeal symptoms at the onset are prominent. There is severe headache, with exquisite tenderness of the limbs and back, the head is retracted, Kernig's sign is present, and there may be photophobia. Such cases may closely resemble other forms of meningitis, but examination of the cerebro-spinal fluid will resolve any doubts, while the presence of flaccid paralysis of one or more limbs is of course valuable evidence in favour of poliomyelitis. In the *Abortive Form* meningeal symptoms are often severe at first, but the disease goes no further, or if there is a temporary weakness of the limbs there is no permanent paralysis.

Spreading Form. Almost always the distribution of the paralysis is at its maximum from the onset, but there are rare cases in which the paralysis begins in the extremities, generally the legs, and during the next day or two ascends to involve the trunk, the intercostal muscles become paralysed, and as the cervical segments of the cord are reached the diaphragm is put out of action, and death is then imminent. The spreading form may however arrest itself before the entire respiratory apparatus is involved, and then after several hours or a day or two, during which time the child may be only kept alive by artificial respiration, strength begins to return to the muscles most recently involved, and recovery may take place. Still more rare is the type which spreads by jumps, that is to say, the level of paralysis may remain stationary for a week or so and then show a sudden advance. Instances have been described in which the paralysis has spread in a downward direction.

Polioencephalitis. Polioencephalitis is very much less common than the various spinal types of poliomyelitis, although the infection is the same. During an epidemic of poliomyelitis not more than about 4 per cent. of the cases will show a cerebral localisation. The symptoms depend on the part of the brain involved, but as a rule the lesion affects one or both hemispheres. The illness has an acute onset and convulsions are at first numerous, but are soon replaced by coma from which the child emerges in a day or so with evidence of paralysis. The distribution is generally that of a spastic hemiplegia, but one limb only may be involved. Blindness from involvement of the occipital cortex, or mental deficiency of varying degree, may also remain after the acute symptoms have passed. The amount of recovery from the paralysis varies, but generally some permanent disability results.

Other clinical types have been described according to the area of brain affected, but they are all rarer than the hemisphere form. Thus in the *cerebellar type* the initial symptoms of illness are rapidly followed by evidence of impaired cerebellar function. The movements of the limbs become inco-ordinate, the gait is ataxic, speech becomes slow and hesitant, and nystagmus is present. The outlook in this type is generally good, a restoration of cerebellar function being gradually regained after several weeks. The name "acute cerebellar ataxy" is often used to designate this group. A *brain stem type* affecting the pons and medulla has been described by Batten. This group is marked by paralysis of one or more of the motor cranial nerves and may involve the oculomotor muscles, facial muscles, or muscles of mastication and deglutition. When the medulla is involved death is likely to be rapid, owing to paralysis of vital centres, but otherwise the brain stem lesions as a rule make a complete recovery.

Diagnosis. Unfortunately it is seldom possible in sporadic cases to make the diagnosis during the pre-paralytic stage, but when there are grounds for suspecting the condition the diagnostic value of examination of the cerebro-spinal fluid has already been emphasised.

Diagnosis is seldom difficult in the spinal form when once the paralysis has made its appearance. Occasionally the pain and tenderness may be so severe as to suggest acute rheumatism, but in poliomyelitis the tenderness is not actually localised to the joints, nor is there any swelling or flushing of the joints, while on the other hand, although movements of the rheumatic limb are painful, there is no actual paralysis. An onset under two years of age may simulate infantile scurvy, but the absence of other scorbutic manifestations such as spongy gums or red cells in the urine differentiates this condition. The meningeal form may closely mimic meningitis, but flaccid paralysis is not a feature of meningitis, and any further doubts may be resolved by examining the cerebro-spinal fluid. The cerebral forms must be distinguished from intracranial tumour by the much more rapid onset and the lack of any extension of the paralysis. Differentiation from encephalitis lethargica may be very difficult ; the occurrence of fits at the onset is in favour of polioencephalitis, but often the distinction remains uncertain until the gradual appearance of some sequela typical of lethargic encephalitis, such for example as Parkinsonism.

Prognosis. Except in the spreading forms, or when the respiratory muscles or medulla are involved, a recovery is usually made from the acute stage of the illness. During epidemics the mortality may rise to 10 per cent.

Certain points must be borne in mind in arriving at a prognosis. The extent of the paralysis is greatest in the early stages, and any change that takes place later will be in the direction of improvement. It is impossible to say just how much recovery will take place, but most of the improvement will come about in the first six months, and muscles that by then have shown no power of recovery are likely to remain permanently paralysed. Recovery begins to appear after a week or two, and some help in formulating a prognosis may be had at that time from the electrical reaction of the muscles. A reaction of degeneration will by then be obtained from those muscles whose nerve supply has been destroyed. Recovery is in some cases complete.

A spread of paralysis has already been mentioned as occurring sometimes in the spinal form. Such recrudescence may take place a week or more after the first attack, and the interval has been as long as twelve weeks ; the relapse may prove fatal. Almost always one attack of poliomyelitis gives lasting immunity, but rare instances of second attacks have been recorded after an interval of two years or more.

Treatment. In the acute stage the patient should be isolated so long as there are catarrhal symptoms, although when once the paralysis has appeared the chance of the patient spreading the infection rapidly diminishes. Those who have been in contact with the case during the pre-paralytic stage should be quarantined and a close watch should be kept on them for the early manifestations of the disease.

When paralysis has developed, the affected limb or limbs should be immobilised by being placed between sandbags, the limb being in such a position as to give a maximum amount of rest to the paralysed muscles. As soon as the acute tenderness has passed off light splints should be applied to keep the limb in the correct position and to prevent contractures. The weight of the bedclothes should be lifted by using a cradle. Careful attention must be directed to keeping the affected limbs warm by the use of loosely knitted woollen stockings or mittens. During the early stages pain may be severe, and can be relieved by giving 5 or 10 grains of aspirin, but in more severe cases small doses of opium (such as Dover's Powder gr. 1 at three years of age) may be required. Relief may also follow the withdrawal of cerebrospinal fluid. Hexamine (gr. 5) should also be given three times a day for a few days in the hope of limiting the extent of the infection.

When the respiratory muscles are involved, as may happen in the spreading variety, artificial respiration may be necessary if life is to be sustained, and it may have to be continued for some days. Under these circumstances a mechanical respirator, such as the Drinker apparatus, is invaluable. Many hospitals are now equipped with these, and as the apparatus is not readily transportable, the patient must be taken to the respirator. Needless to say, constant and skilled nursing is required.

When the acute symptoms have subsided, generally after two or three weeks, light massage of the paralysed muscles should begin, and should be followed by passive movements to prevent contractures. As soon as any voluntary movement can be detected daily active exercises should be encouraged. The length of time for which massage and exercises should be continued will vary with the individual case, but improvement may slowly go on for as long as two years, and therefore these measures should be continued as long as there is any evidence of improvement taking place. In the later stages various orthopædic operations may help to increase the function of the paralysed parts.

Serum Treatment. Several years ago Flexner and Lewis (1910) showed that the serum of patients who were convalescent from poliomyelitis contained a protective substance which could prevent the development of paralysis in animals after they had received inoculations of the virus, and recently this work has been applied to the disease in the human subject. At the present time the evidence as to the value of convalescent serum treatment is conflicting, but several workers [1, 2] have recorded encouraging results. To be effective the serum must be given during the acute febrile stage of the disease and before the appearance of paralysis, and herein lies the importance of the recognition of the pre-paralytic stage. If it is decided to give serum, no time should be lost. The serum should be given intrathecally in an amount slightly less than the amount of cerebro-spinal fluid that has been withdrawn, and a second injection should be given intravenously, the total varying between 25 and 50 c.c. As the serum is human, there is no likelihood of any dangerous reactions. A prophylactic anti-serum prepared from the horse by the Lister Institute of Preventive Medicine is also available and may be given to those who have been exposed to infection, particularly during an epidemic, and for this purpose 5 c.c. should be injected intramuscularly.

ENCEPHALITIS LETHARGICA (EPIDEMIC ENCEPHALITIS)

The first description of encephalitis lethargica was given by Von Economo in 1917, and from then until 1926 the condition assumed epidemic proportions, but since that time the incidence has fallen considerably, although sporadic cases continue to occur. No age is exempt, and although the majority of cases in childhood occur during the later years instances have been recorded in infants of a few weeks old. The causal organism has not yet been definitely identified, but it is probable that the disease is due to a specific filtrable virus, and there is experimental evidence to associate the virus with that of herpes febrilis.

Symptoms. The symptoms fall naturally into two groups, namely, those of the acute stage and those which remain as sequelæ after the acute stage has been survived. At the present day, when cases are mainly sporadic, the acute stage may be easily overlooked and the correct diagnosis may not be reached until one or more of the characteristic sequelæ appear. About a quarter of the cases die in the acute stage, while of those that recover roughly two-thirds are left permanently damaged.

Acute Stage. The onset is generally rapid. The temperature rises three or four degrees, headache and constipation are usual, and there may be nasal catarrh and pains in the back—a state of

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[1] MacNamara, J., *Med. Jour. Australia*, 1928, **2**, 266.

[2] Aycock, Luther and Kramer, *Jour. Amer. Med. Assoc.*, 1929, **92**, 385.

affairs which is easily confused with influenza. After three or four days more characteristic symptoms appear; the child passes into a sleepy state which often appears to be profound, although he may be able to give accurate answers to questions without apparently rousing from his lethargy. In others the stupor passes on to coma, or there may be periods of delirium or hallucinations. Squints and ptosis are common, diplopia may be complained of and there may be facial paralysis or more rarely local paralyses affecting other parts. Difficulty in swallowing is sometimes an early symptom, and occasionally there is persistent hiccough. Coarse tremors of the limbs may be present, and even convulsions may occur, although they are not at all usual. As a rule the fundus oculi shows no changes. The cerebro-spinal fluid shows a lymphocytic reaction during the early stage, the cells numbering from a few up to a hundred or more, and the protein is slightly increased, but the fluid soon returns to normal.

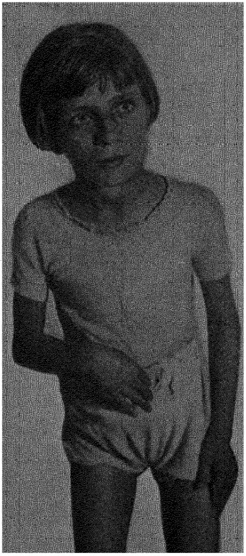
FIG. 87. Child aged ten years with a post-encephalitic Parkinsonian syndrome. Note also the hemiplegic position of the right arm, and the dribble of saliva.

The fever generally lasts about a week, and subsides slowly. Recovery may be complete, but more often a variety of nervous sequelæ remain or may gradually develop several weeks after the initial illness.

Chronic Stage. One of the most common sequelæ is the Parkinsonian syndrome. The symptoms are those of paralysis agitans, and are unmistakable. There is a general increase of muscular rigidity, the face is set and mask-like, the body is bent forward and the legs are slightly bent at the knees. Saliva constantly drools from the mouth, the voice is quiet and monotonous, and the gait is shuffling. Not uncommonly other sequelæ such as mental defect or pyramidal paralysis are associated with the Parkinsonism, while in other cases spastic paralysis may be associated with involuntary movements of a choreiform or athetoid type.

Mental changes occupy a prominent place among the sequelæ. All gradations down to idiocy occur. The whole behaviour often shows a change for the worse, a child who was formerly normal becoming inattentive, spiteful, and heedless of correction. Others take to lying and stealing, and may commit acts of violence. A curious feature sometimes is that the child may sleep during the day and become wide awake at night, disturbing the household by his continuous noisy exuberance. This peculiar disturbance of the sleep rhythm may follow quickly after the early lethargic stage and may last for several months.

Abnormalities of respiration are sometimes a sequel. These generally take the form of bouts of tachypnœa alternating with periods of apnœa, and during the spells of rapid breathing the respirations may be noisy and grunting. Respiratory tics such as frequent yawning or sighing may occur. Oculo-gyric crises occasionally arise, in which the eyes suddenly turn upwards or to one side, remaining fixed in that position for several minutes.

Pathology. The lesions are distributed widely throughout the brain and spinal cord, but there is a special tendency for the nervous tissue of the brain stem and region of the basal ganglia to be involved. Macroscopically in the acute stage the grey matter of the brain shows a characteristic pink flush, while microscopically there is a general increase of small cells throughout the brain, particularly round the small vessels.

Treatment. During the acute stage nursing in a darkened room is needed. There is no specific remedy. Hexamine (gr. 5) can be given orally, and chloral hydrate by mouth or rectum will be indicated if there is acute delirium. The treatment of sequelæ is unsatisfactory. Many drugs have been tried for the Parkinsonian state, but none gives any lasting result. Hyoscine hydrobromide (gr. $\frac{1}{250}$ ter die) by mouth is sometimes useful, and both belladonna and stramonium pushed to the limits of tolerance have seemed beneficial. Paralysis is to be dealt with by suitable splinting, passive and active exercises, and galvanism. Intramuscular injections of sterile milk (1 c.c. twice weekly) is a form of protein-shock treatment which has been recommended for the disorders of sleep ; but for this, as for the other forms of mental disorder, institutional treatment generally becomes necessary.

Encephalo-myelitis following Vaccination and the Acute Exanthemata

It has long been known that encephalitis may be a rare complication of certain of the acute specific fevers, particularly measles, small-pox, chicken-pox, mumps, typhus, and typhoid.

Recently much interest has been taken in this subject because of the increase in the number of cases following vaccination.

These various forms of encephalitis have much in common; the period of incubation between the specific fever and the development of nervous manifestations is about the same in all, and is in the neighbourhood of seven to ten days; the clinical picture is the same; and the pathological appearances are identical. Post-vaccinal encephalo-myelitis will be described as an example.

The onset of post-vaccinal encephalo-myelitis begins about seven to ten days after vaccination, at a time when the vaccine pustules are forming. The temperature rises three or four degrees and the child becomes drowsy and complains of headache or pain down the spine. Vomiting is likely to occur for a day or two, and there may be some rigidity of the neck and stiffness of the spine. The drowsiness passes on to coma, which may steadily deepen to a fatal issue, or a gradual and usually complete recovery may come about after two or three weeks. During the acute stage squints, pyramidal paralysis, or tremors, may occur, and may occasionally persist as permanent sequelæ. Mental defect may also follow. The cerebro-spinal fluid in the acute stage is clear, under increased pressure, and sterile; the cells may be increased from a few up to a hundred or more, and are mostly lymphocytes.

Certain small differences are to be noted in the various forms of encephalitis. The post-vaccinal type may occur after both first and re-vaccinations. It most commonly arises during the years of schooling, and the risk is less when vaccination is carried out in infancy. Of 56 cases collected by Scott, only 7 per cent. occurred under the age of one year. The mortality in the cases following vaccination is as high as 30 per cent., while in the cases following measles it is about 10 per cent., and is lowest in the cases following chicken-pox. The likelihood of encephalitis occurring after any of these illnesses seems to bear no relation to the severity of the initial fever.

Pathology. The lesions are distributed widely throughout the white and grey matter of the brain and spinal cord. There is some vascular engorgement and slight peri-vascular cellular infiltration, but the characteristic feature, and one which distinguishes these cases from polioencephalitis and epidemic encephalitis, is the demyelination of the nerve fibres in the neighbourhood of the small vessels, chiefly in the white matter.

Treatment. Careful nursing and complete rest are essential.

In the post-vaccinal group Horder [1] has recorded a rapid improvement following the intrathecal injection of 5 c.c. of serum obtained from a recently vaccinated patient. Lumbar puncture will be done in the first place for diagnosis, and if it is found to alleviate the symptoms it should be repeated daily during the acute stage.

Spontaneous Encephalo-myelitis

This is a condition which resembles the forms of encephalitis just described, except that the cases arise without preceding cause. Pathologically, there is a type that shows focal demyelination, and clinically resembles cases of encephalitis following vaccination. Another type shows toxic degeneration of nerve cells, often with a slight lymphocytic reaction in the cerebrospinal fluid, and various names have been applied to these cases such as serous meningitis or acute toxic encephalitis. The majority make a complete recovery.

Schilder's Disease (Encephalitis Periaxialis Diffusa)

This rare condition was first described in 1912. Symptoms begin in childhood generally after the third year, and consist of progressive mental degeneration with gradually increasing spastic paralysis and progressive deafness and blindness. The last two symptoms are central in origin, for the ears and eyes are themselves normal. More than one member of a family has been affected. The course is a steadily downhill one, which may be interrupted at times by convulsions. Death takes place after a few years.

Post-mortem examination shows the white matter of the brain to have undergone a patchy greyish degeneration, which microscopically is seen to be due to widespread demyelination of the periaxial sheaths, with secondary fragmentation of the axon and a diffuse increase of neuroglia.

There is no treatment.

CHOREA

(Rheumatic Chorea ; Sydenham's Chorea ; St. Vitus' Dance)

CHOREA is a nervous disorder characterised by :—

(1) Involuntary movements.
(2) Inco-ordination of voluntary movements.
(3) Emotional disturbances.

Etiology. *Age.* Chorea is rarely encountered under five years, that is, before the years of schooling. The youngest case observed by the author was a child two years old, and Poynton has recorded an instance in a baby of only ten months. The incidence rises rapidly after the fifth year, reaching its maximum at about the tenth year, and becomes rare after puberty.

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[1] Horder Sir T., *Lancet*, 1929, *i.*, 1301.

Sex. The sex distribution is unequal, roughly 2 girls being affected for 1 boy.

Type of Child. Chorea is much more common among the children of the poorer classes than among those of the well-to-do. Children who are alert and intelligent, learning quickly at school, are much more likely to be affected than those who are dull and placid, indeed the important part played in the production of chorea by the stress of education, and especially by the anxiety of examinations and scholarships, cannot be doubted. There is often a history of some shock or fright at the beginning of an attack. Thus in one child the fright of a thunderstorm was said to have started the chorea, and in another the movements started within a few hours of witnessing a particularly gruesome street accident. Punishment at school is sometimes cited by the parents as the cause, but it not infrequently proves on investigation that the punishment was given for symptoms which were, in fact, heralding an attack.

Association with Other Rheumatic Manifestations. Chorea is so often associated with other forms of acute rheumatism that it is now widely regarded as the cerebral expression of rheumatic infection, although there is as yet insufficient evidence to place chorea and other forms of rheumatism upon a common pathological basis. If we accept the rheumatic origin of chorea, it is of all forms of acute rheumatism the one most likely to occur alone without other rheumatic manifestations. Thus of 197 choreic children, 52 showed evidence of rheumatic heart disease, and a history of rheumatic polyarthritis occurred in 5 others, so that in all, less than a third showed other forms of rheumatism. Acute rheumatic arthritis and chorea practically never occur simultaneously, which is indeed fortunate, seeing how painful would be the movements of the acutely swollen rheumatic joints.

Pathology. The pathology of chorea is uncertain. The disease is so seldom fatal that opportunities for studying the morbid changes seldom arise, and moreover, fatal cases are generally complicated by rheumatic carditis, which may account to some extent for the vascular changes that have been described. The chief findings have been congestion of the meninges, thrombosis of cortical vessels, and a slight increase of small cells in the brain, often in the region of arterioles and particularly in the neighbourhood of the basal ganglia.

It is to be noted that chorea differs from other inflammatory conditions of the brain in that recovery is invariably complete. Obesity has been recorded as an occasional sequela, but local paralyses or mental defect do not remain. The cerebro-spinal fluid is unaltered, with the exception that Warner[1] has shown the calcium content to be reduced by as much as 15 per cent. The cause of the reduction in calcium is not clear. It is also of interest that the sedimentation rate of the red blood cells, which is increased in acute rheumatic arthritis and carditis, remains unaltered in acute chorea.

The anatomical localisation for the movements of chorea is also uncertain. The striatal region, the corpus luysii, and the region of the red nucleus have all been blamed, while Wilson,[2] from a detailed study of the movements, places the principal lesion in the cortex.

Symptoms. Occasionally the onset of chorea is sufficiently abrupt for a definite date to be fixed to the first symptoms, but more usually the onset is gradual. Often the first symptom to be noticed is that the child continually drops things and becomes fidgety, makes faces, and is easily provoked to tears, or may become dull and unable to concentrate. Symptoms such as these are likely to lead to reprimands at school until it is realised that the child is ill rather than naughty.

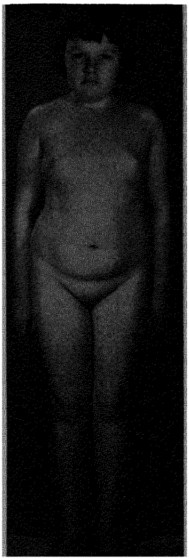

FIG. 88. Obesity after chorea. The patient, a girl aged ten years, became fat after her fourth attack of chorea.

On examination the expression is often dull and heavy, and the mouth may droop, but the movements are the most noticeable feature. These vary very much in degree and are typically haphazard. At one moment the mouth is twisted or the eyebrows raised, a moment later a shoulder is shrugged, a hand jerked or a foot shuffled; in fact, it is impossible to foretell what movement will next appear. The tongue may make clucking noises, and sighing—which is unusual in childhood—occurs because of inco-ordination of respiration. The excursions of the diaphragm are often excessive. The speech becomes hesitating, and words may be clipped short, or spoken in a whisper, or uttered explosively. Rapid alterations between meaningless laughing or crying are common. In a milder case the movements may be brought out by watching the child undress herself, the frantic efforts to undo buttons and the wrestling contortions which the child undergoes leaving no doubt of the diagnosis. The movements of the tongue are uncertain, and if protruded it may be withdrawn very sharply or with a slow deliberation. When the hands are outstretched it is characteristic for the wrist to be flexed and the fingers to be hyper-extended, while the thumb makes constant dipping movements. The tendon reflexes are variable, but in the most severe cases they are generally lost. The knee jerk, when present, may be so altered that one tap to the patella tendon is followed by a series of jerks, until the leg is fully extended.

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[1] Warner, E. C., *Lancet*, 1930, *i.*, 339.

[2] Wilson, S. A. K., "Modern Problems of Neurology," London, 1928.

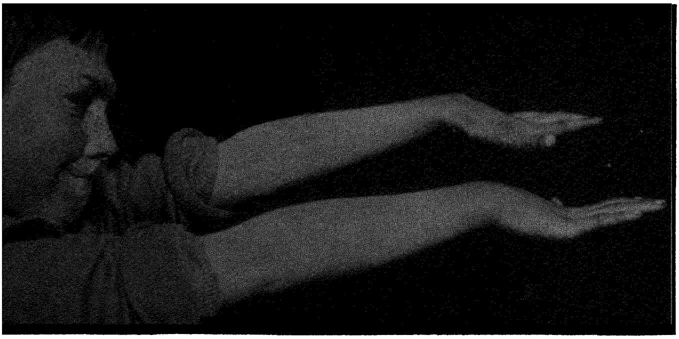

FIG. 89. Choreic hands of a boy nine years old.
Note the facial expression.

Certain particular features call for mention. In the first place, the movements are often more marked on one side of the body than the other, although whether a pure hemi-chorea of rheumatic origin ever occurs is doubtful. The movements cease during sleep, but when they are violent the child may have great difficulty in getting any rest, and there is a real danger of profound exhaustion. Speech may be so severely affected as to be completely lost, and for the space of a month or two the child may be dumb, but the dumbness is never permanent, for the voice returns when the choreic movements cease. Rarely the child becomes more and more excited until acute mania develops ; the behaviour is then very odd, and the child will tear up anything within reach, soil herself, shout, and abuse all and sundry. Eventually a normal mentality is regained, but it may be six months or more before the behaviour settles down. Lastly, there is a limp or paralytic type of chorea. This is the most severe variety, and is invariably accompanied by gross cardiac rheumatism. The child lies in bed unable to raise a limb. The muscles are toneless, and if the limbs are lifted from the bed and released they flop back helplessly. The tendon reflexes are absent. Although the more coarse choreic movements are abolished, if the child is closely watched frequent purposeless movements of the small joints or occasional twitches of the face can be made out. The outlook even in these severest cases depends chiefly upon the condition of the heart, for if the child can survive the carditis then recovery from the chorea can be confidently expected.

In any child with chorea the state of the heart is of the greatest importance. A systolic bruit at the apex is the most common finding and is often accompanied by some dilatation of the heart. In at least a third of these cases the evidence of involvement of the heart clears up during convalescence, and it might be said that the bruit in such cases was functional rather than due to rheumatic inflammation, but it is well known that rheumatic carditis is capable of complete recovery, and, if chorea be accepted as a rheumatic manifestation, it is certainly wiser to treat these bruits with all the respect that rheumatic carditis demands. At other times the occurrence of a diastolic as well as a systolic murmur, or the presence of subcutaneous nodules, or the occasional development of pericarditis, leaves no doubt about the presence of heart disease. On the other hand, there may be nothing to indicate active infection of the heart, and when that is the case the heart action is often noticeably slow and deliberate, the pulse rate dropping to about 60 per minute.

Diagnosis. Chorea in childhood may be regarded almost without exception as rheumatic, but typical choreic movements occasionally arise in the course of other diseases of the brain. Thus encephalitis lethargica may be accompanied by choreic movements, as may a cerebral tumour, and a typical display of chorea may sometimes be seen during the last few days of tuberculous meningitis. Choreic movements may also occur in children with congenital diplegia.

The most common condition with which chorea is confused is habit spasm. Typically a habit spasm consists of one or perhaps two movements which are continually repeated, for example, a child may frequently blink his eyes or purse his mouth, but the movements of chorea are widespread and not repetitive. It may be said that if a child is brought on account of fidgetiness and the mother names one particular movement, it is probable that the child is suffering from a habit spasm rather than chorea.

Course and Prognosis. The prognosis of uncomplicated chorea is good, complete recovery being the rule. Exceptionally chorea is fatal from sheer exhaustion, but practically always when a fatal issue comes about it is owing to coincident carditis. The severity of the movements bears no close relation to the duration of the attack, in fact, often the most troublesome cases to treat are those in which the movements have never been anything but mild. Under treatment the movements disappear, as a rule, in a month to six weeks, but unless convalescence is leisured a relapse is very likely when the child starts again at school. Mention is made elsewhere (p. 564) of those cases of chorea in which the movements have been of the mildest, but have persisted for many months until it has seemed that they have become habitual.

Like all rheumatic conditions, chorea is very likely to relapse, and a child may have as many as five or six attacks. Of 197 children with chorea, 55 experienced one or more relapses.

Treatment. The most important aspect of treatment consists of adequate rest in bed, and in comparison with this, drug therapy is of minor importance. Rest in bed must, however, be absolute; it will not do simply for the child to rest in bed for so many hours a day, spending the remaining hours up and about. The child should be nursed flat, and for the first two or three weeks he should do nothing for himself; for instance, he should not be allowed to feed himself, but should be hand-fed by a nurse. The room should be quiet, and should not be visited by other children who may be in the house. In hospital a quiet corner bed is preferable, but the old practice of putting screens round the bed is not necessary.

The time will come when the child should be allowed to begin to do things for himself and to sit up in bed. This will be when the movements have subsided, and when the child is no longer agitated by trying to perform any simple action. Generally this stage is reached after the third or fourth week, and thereafter simple exercises to encourage co-ordinated movements, such as making jigsaw puzzles or knitting, should be allowed. Massage should also be given as a preliminary to getting up. In the absence of cardiac involvement the child can generally be allowed up in about six weeks, but only if the movements have completely disappeared.

Difficulty often arises over the question of when the choreic child should go back to school. No general rule can be laid down. At the Rheumatism Clinic at The Hospital for Sick Children the average time for which choreic children have been exempt from school has been six months. This has perhaps seemed long in some cases, yet others have relapsed on returning to school after being away for a year. When absence from school implies that the child will spend most of the day in the street, it is better for that child to return to school early, but if there is a garden and the mother or some other responsible person is at home, six months from school should be a minimum. A stay at a convalescent home for three to six months is valuable, particularly if a mild form of education can be combined with the convalescence. This obtains at most of the Heart Homes for children in this country. In many cases half-day attendance at school for a term or two has proved useful, for it may be pointed out that once a choreic child returns to school he soon makes up the ground lost through absence. From what has been said, it will be understood that close co-operation with the School Medical Service and the Education Authorities is essential in the management of these children.

Certain features may call for special treatment. When the movements are so violent that the child is in danger of injuring himself the cot sides should be well padded, and when restlessness makes it difficult for him to get to sleep a warm sponging may do much to promote rest, or in more severe cases a hot pack [1] may be found efficacious in soothing him.

Drugs. There is no specific remedy for chorea, nor has any drug used in chorea been shown capable of preventing that most important complication of all—carditis. Bearing in mind that chorea tends to recover simply with rest in bed, drugs should never be pushed to dangerous limits.

Sedatives may be used when the movements are severe, in order to prevent exhaustion. Chloral, chloretone, phenazone and the bromides are the most useful for this purpose. Chloral (gr. 5 ter die) is useful in promoting sleep, and in the most severe cases as much as gr. 10 at a dose may be needed. When doses of this size are employed, the nurse must be warned to be on the watch for coldness of the hands and feet, a feeble pulse, or undue drowsiness. Chloretone also requires careful supervision, because it is cumulative ; from 3 to 5 grs. may be given three times a day. In mild cases phenazone (gr. 5) combined with an equal amount of bromide may help the child to settle off at night. In the rare cases of maniacal chorea, hyoscine hydrobromide (gr. $\frac{1}{500}$) or morphia (gr. $\frac{1}{24}$) may be necessary. Recently, trial has been made of a drug called "Nirvanol," which belongs to the barbitone group of hypnotics. A course of the drug is given for seven to ten days, and is followed by toxic symptoms of fever, a morbilliform rash, and eosinophilia. Although the choreic movements generally cease with the appearance of the toxic symptoms, the drug is in no sense anti-rheumatic, and is not without danger for its use may be followed by prolonged mental confusion, in fact, Nirvanol may be taken as a good example of how chorea should *not* be treated. Sodium salicylate and aspirin, in view of their known value in relieving the pain of acute rheumatism, have been given an extensive trial in the treatment of chorea, but it cannot be said that they diminish the duration of an attack, although they may do something to lessen the likelihood of other rheumatic complications. Salicylate of soda may be given in doses of 5 to 10 grains three times a day, with double the amount of sodium bicarbonate. Another drug which has been much used is arsenic ; although it has not seemed helpful during the acute stages it is sometimes of value during convalescence, when it may be given as Fowler's solution (m. $2\frac{1}{2}$ ter die). Much larger doses have at times been tried, but their value is very doubtful, while the prospect of toxic symptoms renders their use undesirable.

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[1] To give a hot pack, wrap the child in a blanket wrung out of warm water at 105° F. with a dry blanket outside. The child should lie in the pack for half an hour, and then be dried and have a warm night suit put on.

PINK DISEASE (Erythrœdema Polyneuritica ; Acrodynia)

This is a disease of young children, and is characterised by extreme irritability and misery, pinkness of the extremities, and a variety of sensory, motor and trophic nervous disturbances. The disease was first described by Swift [1] in Australia in 1914, and since then many hundreds of cases have been seen in this country and abroad. The cause is unknown, and at the present time the two principal views are that the disease is either due to an unknown infection or else to some dietetic factor. Points of clinical similarity have been noted between pink disease and pellagra, which is now known to be a deficiency disease, and

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[1] Swift, H., Australasian Medical Congress. Quoted by Wood, *Med. Jour. Australia*, 1921, 1, 145.

Findlay and Stern [1] have produced in rats a condition analogous to pink disease by means of a diet in which the sole source of protein was egg-white.

Etiology. Pink disease is confined to the early years of life between three months and seven years, the majority beginning between six months and two years. The oldest case seen by the author was a girl aged five years and ten months. The sexes are equally affected. Breast-fed infants seem to be just as likely to be affected as those artificially reared, which is a strong point against a vitamin-deficiency theory, and enquiry in older children generally shows no obvious dietetic deficiency. Not infrequently there is a history of some recent infection such as measles or an acute respiratory catarrh. Many cases arise sporadically, but the disease may also occur in local epidemics.

Symptoms. The symptoms form a very characteristic clinical picture. The story generally begins with the ordinary symptoms of an upper respiratory tract catarrh, discharge from the nose, or bronchitis, to which the tell-tale symptoms of the disease are gradually added, so that some few weeks may elapse before the complete picture develops.

The general attitude of the children is often striking. They are miserable, fretful, and restless, and owing to their dislike of the light they prefer to lie with the face burrowed into the pillow and may insist on sleeping in this position, and if they are turned over they will screw up their eyes to avoid the light, or they may lean forward, burying the face between the legs. The appetite is much impaired, and the weight falls until, in severe cases, quite a considerable degree of wasting takes place.

The disease takes its name from the pink appearance of the extremities. The hands and feet, and to a less extent the ears and the tip of the nose, become a bright pinkish-red colour, and are cold and clammy and often look slightly swollen, although there is no actual pitting œdema. The pink colour varies both in extent and duration, and it may fade right away for a few days at a time. Another constant feature is excessive sweating, so much so that the skin over the hands and feet and especially between the fingers and toes becomes pappy and may desquamate in large white flakes, and a sweat rash, consisting of pin-point red papules often accompanied by a fine circular peeling, appears over the wrists and ankles, and may spread up the limbs or may cover the trunk. The scalp is often so irritating that the child continually pulls out handfuls of hair until he may make himself bald; trophic lesions of the hands and feet are shown by a tendency to whitlows, and sometimes by the nails being shed; older children may complain of tingling and numbness, and there may be areas of complete anæsthesia; and stomatitis and gingivitis may reach so severe a degree that the gums gape widely enough for the teeth to be literally picked out of the mouth.

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[1] Findlay and Stern, *Arch. Dis. Child.*, 1929, **4**, 1.

Another constant feature is muscular hypotonia, but without any actual paralysis. The tendon reflexes are usually diminished or may be absent. Almost always there is considerable tachycardia, the pulse rising to as much as 160 per minute, and the blood pressure is sometimes slightly raised.

Diagnosis. When once the condition has been seen, other cases are not likely to be overlooked. The characteristic restlessness and misery of the child, the photophobia, the pink and cold extremities, combined with a sweat rash, considerable hypotonia, and tachycardia, make a picture which is quite unlike any other disease.

Course and Prognosis. The mortality has been estimated at about 5 per cent. Broncho-pneumonia is the most serious complication and is the usual cause of death, although some cases terminate suddenly from cardiac failure. The outlook is very grave when the disease has reached such a state that the teeth are being shed. When recovery takes place it is complete and lasting, and second attacks are unknown. Before recovery takes place, however, the symptoms may drag on for several weeks or even months, during which time their severity shows much variation, so much so that when recovery eventually occurs it is difficult to attribute the result to any particular therapeutic measure.

Pathology. Diffuse changes in the nerves have been found in fatal cases. Myelin degeneration has been noted in the peripheral nerves and also in the vagus and sympathetic. Diffuse infiltration of small round cells in the spinal cord has also been recorded. Sections of the skin show hyperkeratosis.

Treatment. There is no specific remedy, and treatment is chiefly symptomatic. Skilled nursing is needed, at any rate for severe cases. Clothing should be light and of cotton material, and the hands and feet should be protected by cotton gloves and stockings. The diet should be as mixed and varied as the age and appetite will allow, and care should be taken that an adequate amount of the various vitamins is given. Liver has been thought advantageous, and is certainly worth a trial. It is best given as a liver broth[1]. Other cases have seemed to improve after receiving vitamin B in the form of Marmite or fresh brewer's yeast. Drugs may be employed when there is restlessness and loss of sleep, chloral or phenazone (allowing gr. 1 of each per year) being most suitable. For the irritation of the skin alkaline baths are soothing, or a cooling lotion such as lotio calaminæ or lotio hydrarg. perchlor. may be used. Tonics during the active stage of the disease as well as during convalescence are useful, particularly small doses of nux vomica. Ultra-violet light treatment has been recommended, although when there is much photophobia the eyes must be very carefully protected.

Myelitis

Apart from compression myelitis secondary to tuberculous caries of the spine, myelitis is a rare disease in childhood. It may arise after the infectious diseases, or may come on without preceding illness.

The onset may be acute, with fever and pain in the back, and the distribution may be either diffuse, or localised as in transverse myelitis. The symptoms and signs do not differ from those met with in adults. In transverse myelitis there is paralysis and anæsthesia below the level of the lesion with loss of sphincter control, and a zone of hyperæsthesia at the level of the disease. When the lesion is in the lumbar region the legs remain flaccid and there is incontinence of urine and fæces ; when the lesion is situated higher up the cord the legs are spastic and there is retention of urine. In all cases, bed-sores and broncho-pneumonia are dangerous complications.

Diagnosis. Myelitis in children should always lead to a careful examination for spinal caries. In cervical cases the nervous signs may precede any deformity of the cervical vertebræ, and the tuberculous origin of the condition is thus likely to escape detection. The loss of sensation and the involvement of the sphincters will distinguish cases of myelitis from infantile paralysis.

Prognosis and Treatment. Death may occur in the acute stage owing to involvement of the respiratory muscles or from broncho-pneumonia, bed-sores, and exhaustion. When the early illness is survived, paralysis of the limbs and weakness of the sphincters is likely to persist, and complete recovery is exceptional. There is no specific remedy. Expert nursing is essential. Hexamine should be pushed in the early stages, and large doses of potassium iodide have been recommended. When myelitis is secondary to tuberculous disease of the spine treatment must be directed to the latter condition.

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[1] Liver may be conveniently given as a broth. *To prepare Liver Broth.* The liver should be cut into pieces, just covered with water, and simmered for half an hour, any scum should then be removed and the liver be pounded or rubbed through a wire sieve, and finally simmered again for half an hour. The broth may be thickened by adding a little sago.

Spina Bifida

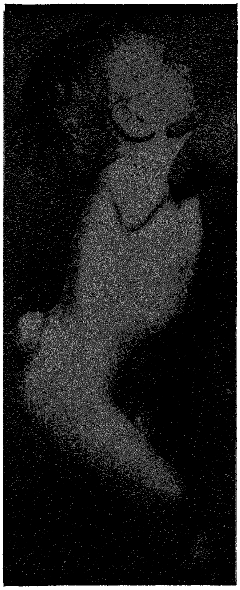

FIG. 90. Spina bifida and meningocele in a boy aged two and a half years.

This is a congenital deformity due to a failure in the fusion of the laminæ of one or more vertebræ, and is usually accompanied by protrusion of the contents of the spinal canal to form a fluid tumour (meningocele).

The tumour may be situated anywhere along the vertebral column, but is most common in the lumbo-sacral region. It is present at birth, and varies in size up to several inches in diameter. The swelling is soft, fluctuant, and may be covered by skin or more often by a thin almost transparent membrane, which readily ulcerates, thereby allowing infection to reach the spinal meninges.

Occasionally cases occur in which no tumour can be seen, although the vertebral canal remains open as may be demonstrated by X-ray examination. Such cases are called " spina bifida occulta," although the site of the deformity is sometimes indicated by dimpling of the skin or by an overlying hairy mole.

In the simplest variety (meningocele) the tumour consists mainly of the meninges and cerebro-spinal fluid, and in these cases there may be no nerve involvement. In the more serious variety (meningo-myelocele) the cord and nerve roots also pass out into the tumour, and the stretching of the nerve tissue is likely to give rise to paralysis of the legs, often with bilateral pes cavus, and paralysis of the bladder and rectum. Occasionally enuresis may be the only clinical evidence of spina bifida occulta, and therefore when a child is brought on account of persistent bed-wetting the possibility of a spinal deformity should be borne in mind.

The outlook for infants with spina bifida is bad. Many die within a few weeks owing to ulceration of the sac and consequent septic meningitis ; in others hydrocephalus appears soon after birth and slowly progresses to a fatal termination.

Treatment. In the absence of any evidence of involvement of nervous tissue or of hydrocephalus, excision of the sac occasionally leads to cure, but there is always the risk that the operation may be quickly followed by the development of hydrocephalus. Surgical treatment is contra-indicated if there is any evidence of paralysis, and in the pure meningocele type it is better to wait for several months to be sure that hydrocephalus is not going to appear. During this time the sac should be protected by a large cotton-wool pad, the tumour being dusted with some antiseptic astringent powder such as tannic acid, and a light celluloid shield may be fitted as a protection. Should ulceration appear during this period, operation should not be delayed. In the occult variety, when incontinence is present, successful operation for removal of fibro-fatty masses compressing the nerve roots has been undertaken.

Peripheral Neuritis

Apart from neuritis following diphtheria, instances of polyneuritis in childhood are rare. Examples of neuritis from lead poisoning occasionally occur as a result of children sucking toys painted with lead paint, and recently a series of 298 infants suffering from lead poisoning has been reported from Japan,[1] the condition being caused by the white face powder containing lead used by the mothers. Paralysis only occurred in 2 per cent. of these infants, but the mortality reached 58 per cent. Arsenical neuritis may also occur from the prolonged administration of the drug, and instances of alcoholic neuritis in children have been recorded. Holt's case occurred in a boy of three years old after drinking twelve ounces of whisky. Peripheral neuritis is also a rare complication of the infectious fevers, and lastly there is a group which arises for no obvious cause—acute toxic polyneuritis.

The symptoms are the same as in adults, and consist usually of a subacute onset, with motor weakness progressing to paralysis. Weakness of the extensor muscles of the hands and feet leads to wrist-drop and foot-drop, the tendon reflexes are lost, and there may be considerable wasting of the muscles. Ataxia and tremors are common. Sensory disturbances vary, but there is generally tenderness of the muscles and nerve trunks.

Most cases undergo a slow recovery after an illness which may last several months. Death may come about from bronchopneumonia, or from involvement of the respiratory muscles, or from paralysis of the heart.

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[1] Fukushima and Matsumoto, *Orient. Jour. Dis. Inf.*, 1928, 3, 27.

Treatment. When the cause can be discovered, such as metallic poisoning, the source of the poison must be removed. Complete rest in bed is essential, and the paralysed groups of muscles should be rested by suitable splinting. Pain may be relieved by the local application of heat, and at a later stage massage and electrical treatment should be used. Of drugs, strychnine in small doses is the most useful.

Peripheral Facial Paralysis (Bell's Palsy)

This is a common condition, thought to be due to compression of the facial nerve by inflammatory œdema of the nerve sheath as it emerges at the stylo-mastoid foramen. Occasionally there is a history of the child being exposed to cold, as in riding in an open car or sitting in a draught. Facial paralysis due to injury at birth, and those cases which arise from middle ear disease, should not be confused with Bell's palsy.

Symptoms. As a rule all the branches of the facial nerve are involved. The normal wrinkles on the affected side of the face are smoothed out, the eye cannot be properly shut, the cheek puffs out when the child is asked to blow, and on showing the teeth the corner of the mouth is drawn to the unaffected side.

The diagnosis of facial paralysis is generally obvious at a glance, and it remains to exclude such causes as middle ear inflammation or intracranial disease. Recovery usually comes about after a few weeks.

Treatment. Rest in bed is necessary at first. The child must be protected from draughts, and the eyes should be bathed three or four times a day with a weak boracic solution as a precautionary measure against conjunctivitis. Inunction of mercury ointment over the stylomastoid foramen is beneficial, and should be combined with massage and electrical treatment.

Progressive Interstitial Hypertrophic Neuritis

This rare condition appears towards the end of childhood, and may affect more than one member of a family. The disease begins gradually and runs a slow course characterised by ataxia and inco-ordinate movements, muscular wasting beginning in the periphery of the limbs, disturbances of sensation, and in some cases by nystagmus and scanning speech. The symptoms bear a close resemblance to Friedreich's ataxy, but the distinguishing feature consists of the palpable thickening of the peripheral and cutaneous nerves. The hypertrophy is painless, and may appear early in the course of the illness.

POST-DIPHTHERITIC PARALYSIS

This is much the most common form of peripheral neuritis in childhood, and its symptoms form so definite a picture as to justify a separate description.

Etiology. Diphtheritic paralysis most commonly arises during the second or third week after diphtheria, but may appear at any time between the first and eighth week. Rolleston has shown that the earlier antitoxin is given in the course of diphtheria, the greater is its effect in preventing paralysis, and certainly at the present day paralysis is most often met with when the initial throat infection has been so mild as to be entirely overlooked, and consequently antitoxin has not been given at all. Antitoxin has no effect on the paralysis itself.

Most cases arise after faucial diphtheria, but by the time the paralysis has appeared diphtheria bacilli can no longer be obtained from the throat, and therefore throat swabs are generally negative. Nevertheless, before admitting a case to a ward containing other children, a preliminary bacteriological examination of the throat should be made.

Symptoms. The onset is subacute, and often the first symptom is a nasal twang to the voice, or difficulty in swallowing or regurgitation of fluids down the nose owing to palatal paralysis. If the child has been up and about he may be noticed to become tired and listless, and unsteady in his gait and hand-actions. The pharynx and œsophagus may later be involved, and the child is then unable to swallow, but chokes and splutters when being fed. Paralysis of accommodation almost always occurs, making it difficult to focus objects close at hand, and so interfering with reading, but external ophthalmoplegia, as shown by squints or ptosis, is unusual. Within a few days the neuritis may become widespread, and leads to a loss of the knee jerks and other tendon reflexes. The face or tongue may become paralysed, and there may be weakness of the limbs, but there is no wasting of muscles, the sphincters are not involved, and generally there is no sensory loss. Involvement of the intercostal muscles and diaphragm may occur, and is always a most dangerous complication; respirations then become rapid and shallow, there is much indrawing of the intercostal spaces, and the child becomes cyanosed and restless, and appears frightened.

Diagnosis. The diagnosis of post-diphtheritic paralysis is not as a rule difficult. The combination of interference with swallowing, a nasal voice, paralysis of accommodation, and loss of tendon reflexes, generally with a history of preceding sore throat, puts the diagnosis beyond doubt.

Course and Prognosis. In 477 cases observed by Rolleston, the mortality was 18 per cent. Respiratory paralysis and pneumonia account for a few, but most of the fatal cases are due to cardiac failure, indeed the prognosis turns largely on the effect of the diphtheritic toxin on the heart. Death from cardiac failure often comes about quite suddenly, and is due at least as much to the acute fatty degeneration of the cardiac muscle as to paralysis of the cardiac nerves. The two chief symptoms of myocarditis are pallor and vomiting, and the presence of these must always make the prognosis very guarded. Tachycardia is usual, the pulse rate running up to 160 or more per minute, and there may be various irregularities of rhythm. Percussion shows that the heart is dilated, and on auscultation the first sound is short and sharp, and systole and diastole tend to become equal in length. Tic-tac heart sounds are of grave significance.

In mild cases recovery begins after two or three weeks. The first muscles to recover are those of respiration, and these are followed by the palatal and eye muscles. The return of the knee jerks is a sure sign of recovery, but sometimes they may remain absent long after the other signs have cleared up. Tachycardia and breathlessness on exertion may also remain for some months, and will inevitably prolong the period of convalescence. Even in the most severe cases, when recovery takes place it is complete, and there are no permanent nervous sequelæ.

Treatment. Skilled nursing is of the greatest importance. The child must be nursed flat in bed with a small pillow, and must on no account be allowed to sit up, since the exertion of this may be sufficient to turn the scale against the weakening myocardium. The diet must be a light one, but when paralysis of the palate makes it difficult for the child to swallow simple milky fluids, they should be thickened to a thick gruel with cornflour, ground rice, arrowroot, etc. In more severe cases, when the child is quite unable to swallow, food should be given through an œsophageal tube. The bowels should be dealt with by daily enemata in preference to drugs.

When the respiratory muscles are involved the outlook is exceedingly grave, and there is then every advantage in putting the child in a mechanical respirator, such as the Drinker apparatus. In this way breathing may be maintained for days, or even weeks, until normal breathing eventually returns.

During the acute stage the two most valuable drugs are strychnine and adrenalin. In severe cases they should be administered hypodermically every four hours, the dose of strychnine being between gr. $\frac{1}{100}$ and $\frac{1}{250}$ according to the age, and the dose of adrenalin between 5 and 15 minims, the size of the dose being regulated more by the severity of the disease than by the age of the child. Atropine gr. $\frac{1}{200}$ every four hours has also been recommended. Brandy may sometimes be used as a temporary stimulant, but its effect is uncertain and it cannot take the place of the previous drugs. It has already been pointed out that antitoxic serum is without effect on the paralysis and therefore should not be given. When the convalescent stage is reached, massage and electrical stimulation to the muscles should be ordered.

It is often difficult to decide when to let the child begin to get up ; if doubt arises it is wiser to err on the safe side and to delay convalescence. It is generally safe to allow the child up when the knee jerks begin to return, but sometimes the return of these is delayed for months and one must then be guided by the condition of the heart. The child should not get up until the first sound at the apex has improved in length and strength, nor until the pulse rate is normal.

Neurofibromatosis (Von Recklinghausen's Disease)

This rare condition affects both children and adults, and may occur in more than one member of a family. The condition may be present at birth, but more usually arises in the later years of childhood. The symptoms are threefold :—

(1) Pigmentation of the Skin. The pigmentation is brown in colour and appears in patches varying from a few millimetres to several inches in diameter, or there may be a profuse brown stippling. It occurs on the limbs, face, and trunk.

(2) Numerous painless pedunculated or sessile pigmented fibromata attached to the skin. In some cases these number several hundred.

(3) Multiple tumours distributed in the course of the peripheral nerves. The tumours vary in size and may be as big as chestnuts. They are generally painless. As a rule there is no interference with the function of the nerves, but when the tumours are

situated within the spinal canal or on the posterior nerve roots severe pain may arise. Histologically the tumours are neurofibromata.

Diagnosis is simple enough when the three cardinal features are present, while the gradual appearance of brown pigmented areas about the skin should suggest the condition. There is no curative treatment, but should there be localising evidence of an intracranial or intraspinal tumour an attempt may be made to remove it by surgical methods.

PROGRESSIVE CEREBRAL DEGENERATIONS

Amaurotic Family Idiocy (Tay-Sach's Disease)

This is a rare condition which affects infants, and is characterised by progressive mental degeneration with spastic paralysis, blindness, and changes in the fundi of the eyes. The disease is almost entirely confined to Jewish children, and several successive members of a family may be affected. At post-mortem examination the brain is harder than usual and the nerve cells are distended with a lipoid substance. A similar degeneration affects the ganglion cells of the retina. The disease is one of a group of metabolic disorders, all of which are characterised by accumulations of lipoid substances (see p. 165).

Symptoms. The infants are born healthy, and development proceeds satisfactorily for about six months. Physical and mental retrogression then appear, and the power to sit up or to support the head is lost. Interest in surrounding objects ceases, and it is gradually borne in on the parents that their child is unable to see. Ultimately complete blindness develops, together with spastic paralysis, and the child becomes bedridden with the muscles in a state of chronic rigidity. Convulsions follow, and progressive emaciation leads to a fatal issue some time during the second year. When vision fails, examination of the eye shows bilateral primary optic atrophy, and at the maculæ there appears the diagnostic "cherry red spot," which is about one-eighth the size of the disc and is surrounded by a pale area of retina.

Treatment is of no avail.

Cerebro-macular Degeneration (Batten)

This is a very rare condition which has been described as the juvenile form of Tay-Sach's disease, but differs in that it is not confined to Jewish children, although it may be familial.

Symptoms are delayed until about the sixth year, when it is noticed that the child is becoming dull, and that the sight is impaired. The symptoms are progressive, mental deterioration becomes profound, blindness is complete, convulsions and widespread spastic paralysis follow, and death ultimately ensues after an illness of several years. Examination of the eyes shows bilateral primary optic atrophy and a characteristic brownish speckling of the retina around the macula. The post-mortem findings are similar to those of amaurotic family idiocy.

Progressive Lenticular Degeneration (Wilson's Disease)

This rare condition is characterised by progressive rigidity of the face, trunk, and limbs, associated with tremors and disturbances of muscle control. The condition is often familial.

The symptoms arise in the later years of childhood or during the second decade. There is a gradually increasing rigidity of the muscles, and a coarse tremor of the limbs which increases when the patient makes any voluntary effort. The mouth is constantly open, saliva dribbles away, and the expression is vacant. Laughing and crying are too easily provoked, and difficulty in speaking and in swallowing are constant features. The rigidity is extra-pyramidal in origin, and the tendon reflexes and plantar responses are normal. Vision is unaffected.

Post-mortem examination shows bilateral degeneration of the lenticular nucleus, associated with multilobular cirrhosis of the liver. The state of the liver does not give rise to symptoms during life.

The course is slowly downhill and is unaffected by treatment. Death ensues after an illness which may last from a few months to a few years.

Friedreich's Ataxy

This is a familial condition, the symptoms depending upon a slowly progressive degeneration of the posterior, lateral, and spino-cerebellar tracts of the spinal cord. The cause is unknown.

Symptoms. The disease begins, as a rule, in the middle years of childhood. The development may previously have been normal, but in some cases walking and talking have been delayed. One of the first symptoms is progressive ataxia, the gait becomes unsteady and staggering, and hand movements become incoordinate. Tremors are likely to be present and are increased during voluntary movements, and there is often nystagmus. The speech becomes slow, monotonous, and scanning. Pes cavus is a constant feature, and there may also be scoliosis. The knee jerks and ankle jerks are lost and the plantar responses may be extensor, but the sphincters are not affected, and ordinary sensation is unimpaired.

The diagnosis should present no difficulty in the face of progressive ataxia with nystagmus, speech changes, absent reflexes, and pes cavus, and perhaps a history of other members of the family being affected. A Wassermann reaction will exclude neurosyphilis. Acute cerebellar ataxies are more rapid in onset, and tend to improve.

The course of Friedreich's ataxy is slowly progressive, but the disease may last for years, and death is generally due to some intercurrent infection. Treatment should be directed to preventing and correcting deformities by splints or other orthopædic measures. There is no curative treatment.

Syringomyelia

This condition generally arises in young adults, but may be met with in the later years of childhood. It consists of a slowly progressive gliosis of the upper part of the spinal cord leading to the formation of cavities in the region of the canalis centralis.

The symptoms in childhood are the same as those in adults and consist of loss of the sensations of heat and pain, beginning in the hands, and associated with wasting of the small muscles and the production of trophic sores and whitlows. Evidence of pyramidal involvement with spastic paralysis of the legs may follow later, or the condition may advance upwards into the medulla, causing weakness of the voice and of the tongue.

The course is slowly progressive over many years. Treatment is confined to the relief of symptoms.

PROGRESSIVE MUSCULAR ATROPHIES

Progressive muscular atrophy in childhood occurs in two clinical forms : progressive spinal muscular atrophy (Werdnig-Hoffmann's paralysis) and amyotonia congenita (Oppenheim's disease).

Progressive Spinal Muscular Atrophy (Werdnig-Hoffmann's Paralysis)

This rare condition is characterised by weakness and wasting of the muscles of the trunk and the proximal parts of the limbs, and is invariably fatal. The disease often affects several members of the family. Both sexes are equally involved.

Symptoms. The onset is insidious. The infant develops normally for the first few months, but at about six months the muscles of the trunk are noticed to be weak, the child is unable to sit up, and soon loses the power even of raising the head off the pillow. It is characteristic that the muscles of the proximal parts of the limbs are involved earlier and to a greater extent than the peripheral muscles, so that the baby cannot lift its arms nor draw up its legs, but lies with the thighs semi-flexed and abducted and the knees bent. Movements of the wrists, fingers and toes are not at first impaired. The limbs are flaccid, and the tendon reflexes are lost. The affected muscles waste considerably, but this may not be at all obvious, as the infant is generally well covered with fat. Sensation is not affected, the muscles are not tender, and there is no impairment of mentality. After a month or two it becomes evident that the intercostal muscles are weakened, for during inspiration the chest wall is indrawn and respiration is almost entirely carried out by the diaphragm, and the cry becomes very weak and feeble. The face muscles and tongue are occasionally involved, and swallowing may be difficult.

The disease is inevitably fatal, generally before the end of the first year. Death is nearly always due to progressive weakness of respiration, with a terminal broncho-pneumonia.

Pathology. The main changes consist of atrophy of the anterior horns of the spinal cord with degeneration and destruction of the anterior horn cells. The anterior nerve roots are shrunken, the muscles show a simple atrophy, and as a rule there is considerable collapse of the lungs.

Diagnosis. Hypotonia with loss of tendon reflexes sometimes accompanies mental defect, and so may be confused with progressive spinal muscular atrophy, but the history of normal development in the early months, together with the involvement of the respiratory muscles and the steady progress of the symptoms, are features that do not occur in amentia. Hypotonia may also be a sequel for some months of any severely debilitating illness in infancy, and is particularly a feature of rickets, but the presence of other rachitic manifestations will prevent confusion.

Treatment. There is no means of avoiding a fatal issue. Skilled nursing is very necessary as the technique of feeding may present difficulties, bed-sores must be prevented, and the final broncho-pneumonia needs trained handling.

Amyotonia Congenita (Oppenheim's Disease)

Amyotonia congenita is generally a milder condition than

Werdnig-Hoffmann's paralysis and affects older children. The pathology of the two conditions is however indistinguishable, and while the familial tendency is not so noticeable in amyotonia congenita, there have been instances of both conditions occurring in the same family. Thus in a family recorded by the author the first child died of amyotonia congenita at five years, the second succumbed to progressive spinal muscular atrophy at twelve months, and the fifth child was seen at three and a half years of age with advanced amyotonia congenita.

Symptoms. The symptoms may be present at birth, or develop soon afterwards. The chief of these is a widespread atony of the muscles. Those of the trunk and proximal parts of the limbs show the greatest weakness, but the distribution is not quite so sharply defined as in Werdnig-Hoffmann's paralysis, and the legs, forearms, and hands are to some extent involved. Owing to the loss of tone, the limbs can easily be put into contortionist attitudes. When the child is held up under the armpits the weakness of the shoulder-girdle muscles is so profound that the arms fly upwards and the child tends to slip through one's hands. The neck is weak and the head cannot be supported steadily on the trunk. The child is unable to raise the hands above the shoulder level, and has to employ both hands to feed itself. A constant feature is the smallness of the muscles, so that the trunk and limbs look unnaturally slender. Sensation is unimpaired, the sphincters are normal, and the mentality is unaffected.

The prognosis is better than in Werdnig-Hoffmann's paralysis, and there is no doubt that many cases show a gradual tendency to improve. The tendon reflexes, which at first are absent, may return and the children may eventually learn to walk, while others adopt various devices for getting about such as rolling over or shuffling along on their buttocks. Those cases that improve to the extent of sitting up are likely to develop severe scoliosis. During the early years however, when the respiratory muscles are weak, lung infections are a constant menace and are very likely to prove fatal. Cases that succumb usually do so during the first two or three years.

Diagnosis. Conditions of severe hypotonicity of muscles are often seen in young children after debilitating illnesses, or may accompany such states of chronic malnutrition as occur in cœliac disease and chronic intestinal indigestion, but in these cases the associated illness will serve to stamp the amyotonia as a secondary feature. The unimpaired mentality is a feature which at once distinguishes Oppenheim's disease from those cases of mental deficiency associated with severe muscular atony.

Treatment. The treatment is unsatisfactory, as there are no curative measures and massage and exercises have little influence. The spinal and chest deformities can to some extent be prevented by the use of a plaster jacket when the children begin to sit up. Every effort should be made to protect these children from respiratory infections. The recent introduction of glycine in the treatment has given promising results. The drug is easily administered in one dose daily of 10 gm., given in a glass of milk. Its expense is a drawback, for it needs to be continued over several months.

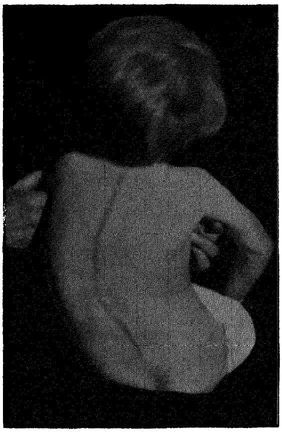

FIG. 91. Child aged two years with amyotonia congenita, showing the spinal curvature.

Peroneal Muscular Atrophy (Charcot-Marie Tooth)

This rare form of muscular atrophy is characterised by an onset in the middle years of childhood and a distribution which commences in the legs and is confined to the distal parts of the limbs. The disease shows both hereditary and familial tendencies.

The first complaint is of weakness of the legs. The peroneal and anterior tibial muscles, together with the small muscles of the feet, undergo a gradual wasting leading to pes cavus, and are replaced by a firm fibrous tissue which allows a surprising amount of function to be retained. Sensation is not affected. The wasting slowly spreads upwards to the lower third of the thigh, and after some years may commence in the muscles of the forearms and hands, giving rise to "claw hands." The disease undergoes a spontaneous arrest at this stage.

There is no treatment.

Migraine

Migraine, or paroxysmal headache, is occasionally met with in childhood. The attacks tend to show a regular periodicity, occurring at intervals of a few weeks. Each attack is localised to some part of the head, often to one side, and usually lasts until nightfall. It is often accompanied by vomiting. Premonitory disturbances of vision are much less common in children than in adults, and ophthalmoplegia, aphasia, and twitching of the face, which may complicate attacks in adults, are practically never met with in childhood.

A relationship between migraine and asthma has already been mentioned (p. 342). Of other causes, eye-strain, and digestive disturbances associated with a too rich or fatty diet, are the most common.

Treatment. During an attack the child should rest in a darkened room. Iced applications to the head are soothing. Aspirin, alone or in combination with phenacetin and caffeine, may be given to relieve the headache.

The prevention of attacks lies in correcting any eye-strain, and modifying the diet on the same lines as for cyclic vomiting (see p. 156). A course of the tincture of gelsemium (m. 5–10 ter die) is sometimes beneficial.

CHAPTER XXI

FUNCTIONAL NERVOUS DISORDERS

Introduction

THE nervous system of the child is much less stable than that of the adult, and a great variety of abnormalities of behaviour is met with throughout the years of childhood. During the early years the nervous system grows proportionately faster than other tissues, and the steady progress from the helpless newborn infant to the child learning to walk, talk, feed itself, play sensibly with its toys, and so forth, calls for the swift expansion of its functions ; and if for any reason the health of the child is interfered with, the effect on the nervous system is likely enough to overshadow the effect on the rest of the body. It should therefore not occasion surprise that in the young child the nervous system may easily be thrown temporarily off its balance, this being shown perhaps by some abnormality of behaviour, or even by a fit, nor that at this age when new associations and ideas are constantly being formed, morbid associations and undesirable habits are easily acquired.

Of the various influences which mould the child's behaviour, one of the most important is the part played by those responsible for the child's training. Some indication of this is shown by the relatively high incidence of disorders of behaviour in only children, who are often affected adversely by the excessive anxiety and fussing of the parents. Children are quick to appreciate the effect of their actions on those around them, and not unnaturally like to occupy the centre of the stage. An example of this is the child who by refusing to eat causes agitation in the nursery, and reaps for himself much misguided coaxing. In this way a child can sometimes attain an amazing ascendancy over parents or nannie, but the fault lies with the adult rather than with the child. At other times, actions which might at first have been easily corrected become transformed into persistent habits owing to the too obvious distress shown by the parents. Even greater harm may be done by playing on the child's sense of fear. This is often done unintentionally, by allowing story books and fairy tales recounting horrors of one sort or another, depicted by suitably gruesome illustrations. It may also be done intentionally, as, for instance, by threatening dire punishments to be meted out by witches, bogies, and suchlike fantasies of the imagination ;

or, even worse, by creating and playing upon a fear of the dark. The implanting of fear in the child's mind is likely to lead to various disturbances of health, accounting perhaps for disordered sleep, and giving rise to morbid fears in the day time.

The behaviour of the nervous system is also closely linked with the health of the rest of the body, and particularly with the digestive system. Such common disorders as chronic intestinal indigestion or constipation are often accompanied by a variety of nervous symptoms such as disturbances of sleep, enuresis, morbid habits like dirt-eating, and teeth grinding, or there may be attacks of sudden pallor due to vasomotor instability.

Certain children are inherently more prone to functional nervous disorders than are others. They form a group of " nervous children," using the word " nervous " in a wider sense than that ordinarily indicated by timidity or fear. These children often come of nervous stock, and the quick speech and restless atmosphere of the parents is reflected in the behaviour of their offspring. Physically, these children are generally wiry and strong enough, although of spare build ; they are active and inquisitive, bursting with energy, and cram a great deal more into their waking hours than do their brothers and sisters. They form habits easily, and are very sensitive to the atmosphere of their environment. There seems no doubt too that these children, with their ceaseless activities, easily deplete their store of glycogen, which lowers their metabolic tolerance for fat, and renders them unduly prone to the various disturbances of health associated with ketosis. When dealing with the functional nervous disturbances of childhood these nervous children must be recognised and their particular metabolic needs must receive attention by limiting their intake of fat and ensuring a sufficiency of carbohydrate.

Head-Banging and Head-Rolling

The habit of rolling the head from side to side is met with chiefly during the first two years. As a rule the infant does not seem to derive any particular pleasure from the habit, and will stop if its attention is attracted, but occasionally the movement is preceded by a cry as though it were being used to soothe a pain. Head-banging occurs at the same age as head-rolling. The infant may bang his head with his fists, or may knock it against some hard object such as the bars of his cot. The banging is sometimes quite vigorous, and the infant may even bruise himself.

The same causes underlie head-banging and head-rolling. Occasionally there is evidence of rickets, although it is doubtful whether rickets alone will account for the movements. More often they arise as a response to some pain or irritation in the head. They are sometimes due to teething, and may occur during the three or four weeks that precede the eruption of a tooth. Another not uncommon cause is earache. The movements may also occur in mentally deficient infants, but even so they are more likely to be an indication of local pain than merely the result of the mental defect.

Treatment consists of relieving the source of irritation, whatever it may be. Small doses of chloral (gr. ½ to 1) should be given three or four times a day to soothe the child.

Thumb-Sucking

This is a common habit, and in some cases goes on practically throughout the day, and even during light sleep, but is most frequent after a meal, or when the baby is tired and sleepy. At times the sucking is so vigorous as to raise a blister or even a corn at the base of the thumb. It is very doubtful if the habit is actually harmful. Certainly there is no good evidence that it will cause deformity of the jaw or protrusion of the teeth, which is a popular fear, but it may lead to a good deal of air swallowing.

As a rule the habit ceases during the second year when the child's interest is taken up with other things, but it occasionally persists for some years. One child only relinquished the habit at five years old when she shed her first tooth, and, thinking that this was caused by her thumb sucking, she feared to lose her remaining teeth.

The habit should be checked in its early stages by continually taking the thumb out of the mouth. Splinting the infant's arms has been advised, but the habit does so little harm that limitation of the child's movements seems scarcely justifiable. Long sleeves which can be buttoned across the hands are sometimes successful. For older children the nails may be painted with a stiff paste of aloes or quinine. The following paste has proved effective; quinine hydrochloride gr. 12; pulv. acacia gr. 12; muc. tragacanth to two drachms.

Spasmus Nutans

This rare condition occurs in the first two years, and consists of nodding of the head associated with nystagmus.

The cause is not definitely known. Most of the cases show some evidence of rickets, and, like rickets, spasmus nutans has a definite seasonal prevalence in the winter months. It has been thought to be due to the infant being reared indoors in a poor light, which incidentally would account for its association with rickets. The nodding of the head only occurs when the child is sitting up, and is thus distinguishable from head rolling. The nods are slow—about thirty a minute—and tend to occur in periods lasting for a few seconds. Often the movements are intensified if the child's attention is fixed on some object. Generally the nodding is from side to side, but may be backwards and forwards, and the nystagmus may be either horizontal, vertical, or rotary, the movements being rapid. A curious feature pointed out by Still is that the nystagmus may be more marked on one side, or even confined to one eye. The children may also show a peculiar way of looking at objects out of the corner of their eyes.

The prognosis is good, the condition clearing up in from a few weeks to a few months. Treatment consists of providing for the child an adequate amount of light by having him out of doors for several hours each day. Exposures to ultra-violet light and the administration of cod-liver oil will be indicated when there is evidence of rickets.

Refusal of Food

Refusal of food is a common complaint during the second year, when the child is getting on to solid food. In infants who are still being reared on the bottle a sudden refusal to suck is generally due to some local condition in the mouth, such as stomatitis or tenderness of the gums from teething, which makes the act of sucking painful.

Refusal of food after the period of weaning is generally the result of faulty management. Meal times should become just as much a part of a child's routine as his bath or his bed-time, and he should not be given the opportunity of thinking that his appetite is a matter of interest. Young children understand a great deal more of what is said about them than is generally realised, and they are also very open to suggestion. The mother who constantly speaks in front of her child of his bad appetite or capricious tastes, and who shows obvious anxiety at his meal times, is making her task unnecessarily difficult by allowing him to see that his behaviour is causing concern. It should be unnecessary to coax a child to eat, and it is even worse to cajole him by the promise of some special tit-bit if he will finish his meal, since this is a direct encouragement to him to be difficult. One often hears accounts of how an exasperated parent has attempted to force a child to eat by putting the food into his mouth, but the attempt is usually futile as the child counters it by vomiting. Another difficult little person is the child who holds food in his mouth for half an hour or more, refusing to swallow it and eventually spitting it out, and neither threats nor beseechings alter his determination.

The prevention of these disorders lies in proper handling from the beginning. It is important to see that the meals are given at regular times, and that nothing is allowed between them. The diet must be a plain and wholesome one, and the child should not be allowed to pick and choose only the sweetest—and often the least digestible—articles on the table. Generally speaking, there should be a healthy disregard of the child's appetite, and if one meal is refused nothing else should be offered until the next meal. Mothers often make the mistake of keeping the milk ration too high in order to safeguard the child's nutrition ; after one year of age a pint of milk a day, including that used in puddings, is ample, and it is often the case that by cutting down the amount of fluid the appetite for solids is improved. There is of course no doubt that many children have a real dislike of certain articles of diet, particularly the fat of meat and green vegetables. Dislikes of this sort should be appreciated and such offending items should not be offered, for the child's taste for them will then develop all the sooner, and meanwhile their value in the diet can easily be replaced by broths, other vegetables, or fruit. Some children have considerable difficulty in learning how to swallow solids, in which case a trial should be made of feeding with cereal-milk feeds thickened to the consistency of a stiff paste.

The treatment of children who are difficult at meal times is often tedious, and in the more severe cases it is generally wise to secure the services of a trained and understanding nurse who can relieve the parents of all their responsibility at meal times.

Anorexia Nervosa, a condition usually met with in young adults, occasionally occurs in other children. The disorder amounts to a self-imposed starvation, and leads to considerable and even dangerous emaciation. In spite of the wasting, the patient denies ill-health, and may stubbornly insist on leading an active life. Psychological conflicts lie at the root of the trouble ; home life or life at school may be maladjusted, and with some the disorder starts from a desire to get rid of an obesity which has caused comment. As a rule treatment at home is a complete failure, and successful management depends on getting these children away for several weeks to a convalescent home where they have the company of other children, and where they are in the hands of sympathetic but firm and persuasive nurses.

Pica, or Dirt-eating

This curious habit is generally met with in children under six years of age. The story is that although the child's appetite for ordinary food is poor, he has a morbid craving for such undesirable articles as wallpaper, coal, hair, earth, or raw vegetables. Almost always examination reveals evidence of chronic intestinal indigestion, such as a pasty complexion, furred tongue, unpleasant breath, abdominal distension, and the passage in the stools of undigested food with an excess of mucus. There may, too, be nervous disturbances such as outbursts of temper, night terrors, or enuresis. The perversion of appetite is as often the result of chronic indigestion as it is the cause ; at all events a history of indigestion often precedes the onset of dirt-eating, and the habit is seldom cured until the digestion has been improved.

Treatment consists of having the child under such close observation that he has no opportunity of indulging his strange tastes, and this will generally require the services of an energetic nannie. The treatment of the digestive state will follow the lines already laid down on p. 199, and should include a diet which will leave but little residue in the bowel, combined with an alkaline tonic before meals, such as a rhubarb and soda mixture. A change of air and surroundings is also of much value.

Nail Biting

This is another common habit, and is important inasmuch as it may help to maintain threadworm infection. The habit should be checked by painting the nails with quinine or aloes paste, or the dentist may be asked to fit a pad to the molar teeth so as to prevent the jaws from being accurately opposed, without which nail biting cannot be successfully performed.

DISTURBANCES OF SLEEP

Sleeplessness

Sleeplessness is a fairly common complaint in children of all ages. In infancy it is usually associated with symptoms of underfeeding or indigestion ; it must also be remembered that at that age earache is particularly likely to give rise to screaming and restlessness at night-time. The irritation of teething also accounts sometimes for restlessness at night.

In the older child various factors contribute to sleeplessness. Faulty training is often to blame. One cannot expect a young child to get to sleep quickly if the hour before bed-time has been spent in riotous and exciting games. On the other hand, an excess of sympathy for the child who cannot get to sleep may easily lead him to think that it is worth his while to stay awake—particularly if he can persuade his parent to lie down with him, or better still if by keeping awake he will be transferred to his parent's bed. Many small children will only sleep when they take to bed with them some special fancy such as an old dolly or a treasured teddy bear, and if these whims make for sleep they should be allowed. Other children beg to have their door slightly ajar, and, so long as they are not in a draught, this should always be allowed. Fear of the dark is easily implanted by a refusal to meet this simple request.

The general health and hygiene of the child are also important factors. Bedclothes should be light, and large enough to be tucked in comfortably. The sleeping suit should preferably be made of some open cellular material, and for the healthy child a vest at night is unnecessary. In older children big suppers at bed-time often account for much disturbance of sleep. Chocolates and sweets or biscuits at bed-time should not be allowed, but if the child is thirsty a small cup of milk and water can be given. Such disturbances of health as constipation or chronic indigestion may be an underlying factor, and enlarged adenoids may cause much restlessness at night by obstructing breathing.

The management of a child's sleep on the lines indicated above removes the need for giving sedatives. When sleeplessness has become so serious as to interfere with health, a dose of chloral at bed-time can be used for a few nights, but there must also be a thorough overhaul of the child's management.

Night Terrors

Night terrors are not uncommon in little children between the ages of two to six years. About an hour or so after the child has dropped asleep he sits up in bed crying and screaming or muttering to himself with obvious fright. During the attack the child stares ahead with unseeing eyes, and it may be several minutes before the mother or nannie is able to calm him or make him recognise her. As the attack subsides the child will generally allow himself to be soothed back to sleep, and remembers nothing of it next day. Occasionally the attack is repeated more than once during the night, or they may recur for several nights in succession or only at long intervals. Rarely, similar attacks occur during the day-time. Holt has pointed out that these attacks are often followed by the passage of a large amount of pale urine.

Various causes contribute to night terrors. From the words which the child lets fall it may be evident that he is living over again some action of the previous day, the action being twisted and misshapen into nightmare proportions. As a rule there is some disturbance of health underlying the attacks, such as overloading the stomach at bed-time, indigestion, constipation, or naso-pharyngeal obstruction, and these must be reckoned with when advising treatment. Other treatment should consist of a course of sedative drugs at night-time, and there is nothing better than small doses of chloral and bromide—gr. 2½ of each for a child of four or five years. If it can be arranged, it is as well for some adult to sleep in the child's room for a few nights, and the child should sleep with his door open so that his distress may be quickly heard.

Somnambulism and Talking while Asleep

These are not uncommon in children of school age, and those affected are generally of a nervous type. Often the subject of conversation refers to school work, and the strain of competitive education, particularly of examinations, sometimes seems to be a determining factor. As in other disturbances of sleep, temporary irregularities of the general health may be associated with these symptoms, and must receive appropriate treatment. Small doses of sedative drugs at night-time are indicated. and a rest from schooling is often advisable.

Habit Spasm (Tic)

Habit spasms or tics are one of the most common disorders of childhood. They consist of frequently repeated movements which, although co-ordinated, are purposeless, such as blinking the eyes, twisting the mouth to one side, or tossing the head.

The movements may take a great variety of forms. In roughly half the cases the muscles of the face are involved, the most common movements being of a grimacing sort, such as tightly screwing up the eyes, raising the eyebrows, or pulling the nose and mouth to one side, but other parts of the body may be affected. The arms are more likely to be involved than the legs. Shrugging of the shoulders is a frequent variety, while tricks of the fingers, stamping of the feet, or writhing of the whole body, are a few further examples from the almost innumerable forms which habit spasms may take. As a rule the child has only one habit movement at a time, although after a few days or weeks it may be replaced by another one, but at times a child may have multiple movements, so that during the course of examination several different tics are seen. Tic movements cease during sleep and also if the attention is closely held, and will sometimes disappear so long as the child is under observation. Certain tic movements have been dignified by special names. There are the respiratory tics, in which the child may make frequent efforts to clear the throat, or perhaps is constantly sniffing or grunting. The " tic convulsif " movements are widespread and violent, while the " psychical tics " consist of motor manifestations together with the production of impulsive sounds. In this last form the child may continually repeat obscene words (coprolalia), or may imitate the remarks of those around. Not far removed from these children are those who suffer from obsessions ; for instance, they may feel compelled to walk only upon the divisions between pavement stones, or they must touch every lamp post, or must repeat the same action a certain number of times.

Probably several factors underlie the production of habit spasms. Many of the children are of a highly strung, nervous temperament, which may also be exhibited in other ways, such as stammering and stuttering, or by irregularities of sleep. It may be noted that habit spasms are most common between five and ten years of age, years in which education begins to fill most of the day and often leaves the brain active at night. These are the years, too, when children, having consolidated the rapid growth of infancy, again grow rapidly, and it is in this " leggy " stage that various manifestations of physical and nervous exhaustion are most likely to appear, of which habit spasms may be an example. From the character of the movements it is likely that some of them begin for a definite reason, such as gaping or screwing up the eyes to free a sticky eyelid, or tossing the head to shake the hair from the eyes. It has already been pointed out how often chronic indigestion is accompanied by nervous disturbance, and in children with habit spasm there is not infrequently evidence of a disordered alimentary function.

Diagnosis. The diagnosis is in most cases a simple matter, the essential feature being the repetition of one or more movements, the history often going back for several months. The most important condition from which tics must be distinguished is chorea. The movements of chorea are inco-ordinate, are not confined to one group of muscles, and there is no obvious tendency to repetition of any one movement. Sighing and interference with speech are common in chorea, but not in habit spasm, and the choreic child has difficulty in grasping objects steadily, so that he is constantly dropping things and may be unable to feed himself. These actions are performed normally by a child with habit spasm. Chorea also is more common in girls, while habit spasms affect the sexes equally. Real difficulty arises when the movements of chorea have never been anything but mild, but are said to have persisted for many months. Observation of these children inclines one to think that although the movements may have been truly choreic in the first place, they are no longer so, but have become habitual. Habitual chorea would perhaps be a better term for them than rheumatic chorea, and from the point of view of treatment they should be managed on the lines indicated for habit spasm. Tics must also be distinguished from the rare condition of myoclonus, which consists of the isolated contraction of individual muscles.

Prognosis. Although habit spasms may last for months or years, and occasionally continue into adult life, they do not of themselves interfere with health. Unfortunately it often happens that the cure of one habit spasm is soon followed by the development of another.

Treatment. In the first place the general health must be raised to as high a level as possible, by seeing that the child has a proper allowance of fresh air, daily exercise, and sufficient rest. Such local causes of irritation as eyestrain, dental caries, chronic infection of the tonsils, constipation, or indigestion must be dealt with.

Not uncommonly there are factors in the environment and home management which affect these children adversely, and then little progress will be made until a period of convalescence for two or three months can be arranged in the country or at the seaside. It is of little use sending these children away with their parents, for they need a complete change. Prolonged rest in bed, which is so essential in the treatment of chorea, is unnecessary in habit spasm, and as soon as these children are away they should be allowed to run about out of doors as much as possible. If schooling is thought to be exerting a bad effect, a period of freedom from lessons for a term should be advised, but otherwise, and especially if the child is to be treated at home, schooling should continue, because separation of the child from his school-fellows may leave him with little to interest himself, and the habit may become all the more deeply ingrained. School authorities often fear that other children may copy the movements, but this seldom happens.

Drugs play but a small part. A dose of 5 or 10 drops of Easton's syrup in a teaspoonful of water after meals has sometimes seemed useful, and when the movements are associated with physical and nervous exhaustion a combination of phenazone and arsenic is beneficial. For a child of six years a suitable mixture would be : phenazone gr. 3 ; liquor arsenicalis m. 1½ ; syrup of ginger m. 10 ; chloroform water to 1 teaspoonful.

Disorders of Speech

Most infants can make such simple sounds as " Mum Mum " and " Dad Dad " by the end of the first year, and the use of simple words and the stringing together of two or three words into little sentences is as a rule accomplished towards the end of the second year. Backwardness in talking should not be reckoned with until the child has failed to produce any words at two years of age.

Delay in talking may arise from various causes, but it may be mentioned here that one of the least common is tongue-tie. One of the most frequent causes is deafness, for speech is only acquired after the child has learned to interpret the sound of the spoken word, and absolute deafness will lead to complete dumbness. The lesser degrees of deafness not only cause delay in talking, but may make the child speak his words indistinctly, because he only hears them imperfectly. Thus he may clip off the ends of his words as in saying că for cat, or dŏy for dolly. A history of otorrhœa will at once give a clue to the cause of lateness in talking, or the hearing may be imperfect because of Eustachian obstruction caused by such conditions as nasal catarrh or hypertrophied adenoids. Deafness in a young child may also be of central origin, as for instance after post-basic meningitis, and because the deafness in these cases is generally severe the child is likely to remain mute.

The next most likely cause of delayed speech is mental deficiency. There will generally be other traits by which this may be recognised, such as lateness in walking, difficulty in acquiring habits of cleanliness, or lack of interest in surrounding objects. Some degree of speech is, however, eventually acquired in all but the most severe cases of idiocy. Distortions of speech are likely, and words may be so mispronounced that only those in constant contact with the child can glean any sense from them. Distortion of words is common enough in the early vocabulary of normal children, but this seldom lasts more than a year or two. The mentally defective child may, however, continue his baby language for several years.

Congenital aphasia is fortunately rare, but must be remembered as a possible cause of delayed talking and of unintelligible jargon. The most common form is congenital word-deafness, in which with a normal auditory apparatus the children are unable to interpret what they hear, although, curiously enough, they may be able to understand and be interested in other sounds such as a whistle or a tune. Being unable to understand what is said to them, these children often seem oblivious of remarks made to them, and are likely to be dubbed as mentally defective, the true nature of their condition being overlooked ; actually, however, they are not mentally backward, and by assiduous training directed toward cultivating their visual-perceptive centres instead of their auditory-perceptive centres they can be taught to lip-read, and so to interpret the meaning of what they see.

Congenital word-blindness may also be mentioned here. In this case the child cannot interpret what he sees although he is able to understand the spoken word quite well. Talking is not interfered with, but the child is unable to read. This defect may be mistaken for mental deficiency, or wrongly attributed to some error of refraction. Much may be accomplished for these children by specialised training, making the auditory-perceptive and touch-perceptive centres take over the work of the visual perceptive centres.

Occasionally failure to talk may simply be an oddity of behaviour. The child may then appear to take no notice when he is addressed nor make any effort to answer. He may be like this to strangers—while he will converse normally with his parents. Such children are sometimes precocious in other ways, but more often as they grow up it is realised that they are mentally below the normal and they may be passionate, obstinate, and difficult to handle. Refusal to talk is also part of the stock-in-trade of the "negative" child ; the repeated efforts of the parents to encourage him to speak simply drive him further into a determined silence. Usually these children can speak quite well, and having failed to say anything for several weeks may suddenly astound the household by making some apt remark.

Loss of speech after it has been acquired may be due to various causes. The onset of deafness in the early years, when speech is still a hesitating affair, may cause the child to forget what words he has learned and may lead to dumbness, and special methods of training may then be necessary to re-educate the power of speech. Aphonia is also an occasional manifestation of rheumatic chorea, but the speech invariably returns after a few weeks as the chorea passes off. Instances of complete loss of speech for hours or days after shocks or frights have been recorded in nervous children.

Stuttering (Stammering). This is due to an inco-ordination of the various muscles concerned in speech, and results in a staccato repetition of the first letter or syllable of a word, or at times in a complete failure to get out any sound for a few seconds. The inco-ordination may affect the diaphragm and muscles of respiration, producing an irregular flow of air ; or the muscles of the larynx, palate, tongue, and lips may be involved, leading to difficulty in saying the consonants and vowels produced by these different structures.

Stuttering is more frequent in boys than in girls. It is most common at about six years, at a time when other nervous disturbances are also common, and the children are generally of a highly strung, excitable type. The condition is worse under the stress of nervousness or shyness, while over-eagerness to talk, and talking too rapidly, are often important factors. A rather similar condition is to be seen in younger children when they attempt to talk after a severe bout of crying. Occasionally a child begins to stutter after mimicking another child. Stuttering often clears up spontaneously after two or three weeks, and a child may have several separate bouts of it. If the condition lasts for more than a month, the child should be taken properly in hand in order to get rid of the habit as soon as possible.

The first point in the management must be a thorough overhaul of the child's general health, and the daily routine must be modified so as to curtail excitement and minimise fatigue. At times a change at the seaside combined with some tonic may be all that is required. The breathing is so often at fault that a course of breathing exercises is generally invaluable, for the child must be taught to fill his lungs before trying to speak. He must also be taught to speak *slowly and quietly*, and must be given to understand that there is no need for hurry. When the speech breaks down into stammering the child should stop the sentence and be made to pause and breathe slowly before starting off again. It is often a good plan to get these children to read out aloud, or in severe instances to sing their sentences, since singing automatically demands deep and steady breathing. Bad stutterers are often able to sing without difficulty.

Idioglossia. Comment has already been made on the distortion of words in baby language—for instance, a child may pronounce the word garden as " dudden "—and that in mentally defective children this may persist into later childhood. Occasionally it is met with in children who are otherwise very intelligent, and almost every word may be so distorted that the child seems to speak a language of his own. This is termed " idioglossia." These children understand ordinary language perfectly, and although their own speech may be incomprehensible to others, it is sensible enough to them. Guthrie,[1] in the following words, has attributed idioglossia to a peculiar defect of hearing: " The defect seems to be one of audition. Without being deaf in the ordinary sense, the patient is unable to discriminate between sounds. His hearing does not detect any difference between his own jargon and words correctly spoken. In favour of the view that idioglossia is primarily a fault in audition, analogous to colour-blindness, is the fact that in typical cases there is complete absence of an ear for music."

The correction of idioglossia calls for prolonged and special training. A useful method is for the teacher and child to pronounce the words in front of a mirror so that the child may watch himself repeating the movements. By employing the visual centres in this way, with patience and perseverance a proper mode of speech can be inculcated.

Enuresis (Incontinence of Urine)

Incontinence of urine is one of the most common functional disorders of childhood, and probably none causes more worry to the child, the parents, and the doctor. Functional incontinence of urine is, as a rule, confined to the hours of sleep, and those children who also wet themselves by day have generally suffered for a longer time from nocturnal enuresis.

The majority of children learn clean habits before they are two years old, and even if wetting continues after this it is apt to be looked upon as a mere matter of nursery training, so that medical advice is not as a rule sought until after three years of age, and most cases are brought between five and ten years. In fully half of the cases the history shows that the bed-wetting has persisted from infancy. In others, the bed-wetting has dated from a variety of causes of which some, such as the infectious fevers, have lowered the child's health and thereby made nervous disorders of one sort and another more likely, while others, such as frights or injuries, have produced a sufficient psychical commotion to start off the complaint. Fortunately, as self-confidence becomes more established in the later years of childhood, the majority of bed-wetters undergo a spontaneous recovery.

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[1] Leonard Guthrie, " Diseases of Children," by Garrod Batten and Thursfield, 1913, p. 738.

The incidence of enuresis varies with both age and environment. The author's enquiry among several large residential homes for Poor Law children—with a total of over 5,000 children—showed the incidence between the ages of five and ten years to be approximately 5 per cent. Roughly 3 boys were affected for every 2 girls.

When dealing with a child brought on account of enuresis, organic causes for the complaint must first of all be excluded. Enuresis may be an early symptom of diabetes mellitus, and is also likely to be present in chronic interstitial nephritis, and in the rare condition of diabetes insipidus. The characters of the urine, together with a thorough examination of the child, should serve to separate these organic causes. Occasionally incontinence is accompanied by a persistently palpable bladder, which is always sure evidence of some underlying organic condition causing obstruction to the flow from the bladder, such as the presence of mucosal valves in the urethra. Examination should also be made for sacral spina bifida or spina bifida occulta, for these, by causing a neuromuscular inco-ordination at the neck of the bladder, may account for incontinence.

In the functional bed-wetter the urine rarely shows any abnormality to account for the condition. Hyper-acidity of the urine has been blamed, but is more likely to produce frequency of micturition than incontinence, and in any case the excessive acidity can easily be reduced with potassium citrate. In the same way the presence of uric acid or oxalate crystals should be treated by giving alkaline diuretics. The presence of pus in the urine indicates that the incontinence is but a symptom of some underlying organic disease of the urinary tract. It has been shown that in some cases the output of urine during the night exceeds the amount produced during the day, which is, of course, the reverse of normal.

Various factors play their part in contributing to the persistence of bed-wetting. Many of the children are intelligent enough to be made intensely miserable by the repetition of their nightly misfortune, and the reproach of their parents, with alternate scoldings and pleadings for better behaviour, makes the menace of a wet bed something to be dreaded. The child may conceal his wretched feelings by a show of bravado to the whole affair, but his anxiety is none the less real. Punishment for these children is generally worse than useless, for it increases their anxiety over the condition instead of encouraging self-confidence in their ability to keep their bed dry. It is a striking fact how almost invariably the enuresis stops when these children are taken into hospital, although when they return home the wetting only too often starts again.

The incontinent child sometimes exhibits other nervous traits, such as habit spasms, stuttering, and so forth, but many children with this unfortunate habit are in other respects quite normal. As an entirely different proposition one also meets with bed-wetting among children of low intelligence. The early training of these children is more arduous than in normal children, and greater watchfulness is needed to inculcate habits of cleanliness. Some of these children fail to appreciate any discomfort from their wet bed, while others are too lazy to bother about getting out at night.

Various sources of local irritation may play a part in keeping up enuresis. Threadworms are often associated with bed-wetting, although whether they operate merely by producing perineal irritation at night-time seems doubtful, for it is at least as likely that the catarrhal state of the bowel, of which the worms stand as evidence, promotes a state of nervous exhaustion and so makes it more difficult for the child to maintain a proper nervous control of the bladder. Constipation is another condition which often requires attention before the enuresis can be overcome. In other cases septic tonsils and adenoids or the irritation of carious teeth may be contributory factors. Occasionally the history shows that the child is having indigestible suppers or long drinks before going to bed. It has recently been suggested that enuresis may result from the child's hypersensitivity to the material of his bedding, and that good results may follow the substitution of the feather pillow and horsehair mattress by bedding stuffed with a vegetable fibre (Bray). Certainly when enuresis accompanies other allergic conditions such as asthma or hay-fever, improvement in these is often followed by a disappearance of the bed-wetting.

Treatment. Although certain routine measures must be applied to most cases, such as restricting the fluid intake and rousing the child at night to pass water, there is no method which can be entirely relied upon, and each case requires a careful analysis before the exact method of treatment is decided.

Particular care must be given to improving the general health where that is at fault. The daily management of the child must be regularised, meals must be at definite times, and the last meal should be at tea-time. The diet should be an easily digestible one, and fluid should only be taken with meals, no drinks being given after tea. Such local conditions as constipation, threadworms, vulvitis, or balanitis must be treated. It is important to see that the bladder is properly emptied at bed-time. Not infrequently the child is found to be wet within an hour of getting into bed, and in that case he must be encouraged to micturate more effectively at bed-time, and it may be necessary to get him up after half an hour to repeat the process. More often wetting occurs later in the night, and the child should then be lifted out at 10 o'clock. It is important that when the child is lifted he should be thoroughly roused, so as to realise the purpose for which he is being wakened, for if he remains drowsy he may only incompletely evacuate his bladder. Young children should not, as a rule, be roused again, but if the habit persists into late childhood it may be necessary for the child to get up again at about 2 a.m. for a few weeks before the habit can be broken.

There is no doubt that in older children there is a strong psychological side in treatment, and it is essential to get the right atmosphere into the home. Scoldings should be tabooed, and it is equally harmful for the parents frequently to lament over the child's shortcomings in his presence. The attitude should be one of reassurance, and confidence in his ultimate recovery. It is better to praise a dry night and to leave unremarked a failure. So long as a child is made to feel ashamed and inferior to his fellows, which may easily happen at boarding-school, so long will his misfortunes continue. It is often clear that a child has lost confidence in his power to be dry, and the doctor is usually in the best position to restore his confidence by talking to him in a sympathetic and reassuring way. A different attitude may be required for children who are mentally backward. It is often necessary to offer some sort of bribe to these children to make them realise that it is worth their while to try to be dry at night; others will sometimes respond if it is put to them in a competitive way, such as by letting them ring on a calendar the dates of the dry nights.

It is doubtful if simple phimosis is a cause. Enuresis sometimes ceases after circumcision, but the operation is no panacea ; indeed, it often has no effect, and therefore should not be advised unless there is some evidence of preputial irritation. When it has seemed that the capacity of the bladder is unusually small, so that in addition to enuresis the child is frequently passing small amounts of urine during the day, improvement has followed a gradual distention of the bladder with fluid given slowly by catheter.

Various drugs have been recommended, but none are likely to help unless the general management outlined above is adhered to. The most popular is belladonna, but to be useful it must be given in large doses. The initial dose should be m. 5 of the tincture three times daily, and the dose should be increased by m. 2½ every fifth day until as much as thirty or forty drops are being taken, or until the enuresis checks. When that happens the maximum dose should be continued for a fortnight and then be gradually reduced by m. 2½ each week, the whole course taking about three months. Symptoms of intolerance to belladonna, such as flushing of the face, a scarlatiniform rash, dilatation of the pupils and diplopia, or noisy delirium, will call for a reduction in the dose. Another drug which has sometimes seemed useful is ergot, of which the liquid extract may be combined with belladonna, giving the same dose of each, and increasing them both together. Strychnine (two or three drops of the tincture of nux vomica) is also worth a trial in combination with the previous drugs.

Ephedrine hydrochloride is sometimes successful, but there is no means of deciding which children it may help. It should be given at bedtime, beginning with ½ gr. and increasing by ½ gr. every fourth or fifth night until 2 gr. is being taken. If by then the enuresis is not responding, the drug should be discontinued, but should the bed-wetting cease the dose should be gradually reduced in the same way that it was increased. Signs of intolerance, such as excitement or nightmares, would contra-indicate its use.

Treatment with thyroid extract has its advocates, but in the author's experience it has been disappointing. It is likely enough that children who lack thyroid from birth will be late in acquiring control of the bladder, just as they are late in learning other things, but the enuresis is due to their mental backwardness, and is comparable with the enuresis that keeps company with mental defect from other causes. It cannot be said that the majority of enuretic children show other clinical evidence of lack of thyroid; in fact, apart from their enuresis they are usually normal, both mentally and physically.

Diurnal enuresis is generally accompanied by nocturnal bedwetting, but is much less common. It should be treated along the same lines, and in addition it is often a good plan to make the children pass urine at regular intervals during the day. During the first week they should be sent to micturate every hour, in the next week every two hours, and so on, the interval being gradually increased until they can hold their water for four hours at a stretch.

Incontinence of Fæces

Incontinence of fæces may arise from organic diseases affecting the nervous mechanism of the lower bowel, such for example as spina bifida, caries of the lumbar vertebræ, or myelitis. It is also not uncommon in mental deficiency. Incontinence of fæces is, however, more frequently met with without organic cause, and is in fact comparable to functional incontinence of urine, with which it is often associated. It differs, however, in that it occurs more frequently by day than by night, and as a rule does not date from infancy, but is acquired some time later. It may follow debilitating illnesses such as measles or whooping-cough, and it occasionally arises in children with chronic indigestion, the stools being undigested and containing an excess of mucus. Mismanagement at home seems to account for some cases, at all events the trouble often disappears as soon as the child is taken into hospital. In other cases chronic constipation is the underlying factor, the child seeming to be unconscious of what is escaping from the overloaded and distended rectum.

Treatment. The general health needs to be at as high a standard as possible, and the diet must be easily digestible, with avoidance of foods that are likely to leave any bulky residue. Of drugs, opium is by far the most useful, and generally stops the habit in a few days. It may be given as Dover's powder, the dose to a child of six years being 2 gr. twice a day for a few days. Other measures, such as injecting sclerosing fluids into the perianal tissues, or the local use of a cautery, are both painful and unnecessary. When constipation is the cause, it is best dealt with by repeated enemata.

Masturbation

Although masturbation may occur at any age in childhood, it is most often met with in the first two years, and is then more common in girls. In later childhood it becomes more frequent in boys.

In infancy the habit is performed in various ways, usually by crossing the thighs and working them one against the other, or alternately bending and straightening the thighs, or sliding the buttocks forwards and backwards on the edge of a chair, or by rubbing the perineum against any projecting piece of furniture. The hands are not, as a rule, used in the act until some years later. During the performance the infant has an expression of concentration, becomes red in the face, strongly resents any attempt at interference, and afterwards may seem pale and exhausted. It must be remembered that many infants practise other sorts of habitual movements which may at first have the appearance of masturbation, but they are not accompanied by a flushing of the face nor followed by exhaustion, and are, in fact, harmless. For instance, young children will sometimes rock their bodies to and fro for long periods, others when lying down will repeatedly arch the body off the bed. As the infant grows older and finds more and more to occupy his mind these movements tend to disappear.

Masturbation not unnaturally alarms and distresses the parents, for they fear that their child is sexually precocious or in some way abnormal. It should be pointed out to them that this is not the case, and that the habit at that age has no particular moral stigma attaching to it, but is to be regarded in much the same light as other objectionable habits like spitting or nail biting. On the other hand, there is no doubt that the child finds the habit a pleasurable one, and is not likely to abandon it unless active measures are taken.

In undertaking treatment it is essential that the mother should see the habit in its proper light, for the responsibility of checking it will devolve on her. This she must do firmly every time the child tries to perform the act, but without showing apprehension or distress. At first this will inevitably lead to severe tussles, from which the mother must always emerge the victor. Meanwhile, as far as possible, the child's attention should be diverted to toys and other interests. In older children it is of the greatest importance that a frank and open attitude towards the habit should be preserved, otherwise the child is likely to continue the practice in privacy, which at once adds to the difficulty of putting an end to it.

Any source of local irritation must receive attention. The presence of worms should always be suspected, and even if the history of them is denied it is worth while giving a vermifuge.

Constipation is also at times a factor. In little girls there is often some degree of vulvitis, which may be enough to give rise to slight local discharge, or may be indicated by a reddened and sore perineum, and in these circumstances the child should be given warm hip-baths twice a day. One of the best solutions is potassium permanganate, adding enough crystals to the bath to make the water bright pink ; or a weak iodine bath may be used, allowing an ounce of tinct. iodi. to 2 gallons. In boys, the prepuce may be tight and adherent, or there may be some balanitis which would be an indication for circumcision, but unless some such local condition is present the operation should not be undertaken, as it may not be curative, and indeed, by allowing the sensitive glans to be continually exposed, it may cause the habit to become more deeply engrained.

In infants some method of mechanical restraint is often helpful, but should not be used in older children, as it draws too much attention to the condition. Thigh rubbing may be prevented by the wearing of broad garters high up the thigh with a thick pad on the inner side to prevent the thighs from being brought together, or the thighs may be kept apart by a short bar connecting rings round each thigh. A combination of belladonna and the bromides sometimes gives satisfactory results. Liquid extract of salix nigra, a drug which is used as a sexual sedative, is sometimes of help in these cases, and may be given in doses of 5 m. ter die to a child of one or two years.

Hysteria

Hysteria is uncommon in childhood, and seldom occurs under five years of age, but from then onwards the incidence gradually increases. The youngest instance observed by the author was a little girl aged three and a half, who for two months after an attack of jaundice had been unable to walk properly, but dragged the left foot along as though it were a dead thing. This curious gait was somewhat inconstant, and became worse when she was being watched. Persuasion by massage and exercises led to a rapid recovery.

Hysteria in children shows certain differences as compared with adults. The preponderance in females is less striking, although girls are more often affected than boys. The hysterical manifestations are generally more simple in character, and are likely to be confined to one portion of the body, such as an arm or leg, and are for the most part restricted to such motor phenomena as paralysis or abnormal movements. The gait is particularly likely to be affected. Often the onset dates from some illness, or may begin after some shock or fright, while occasionally the hysterical condition is super-added to some organic disease.

The following cases serve to illustrate some of these features.

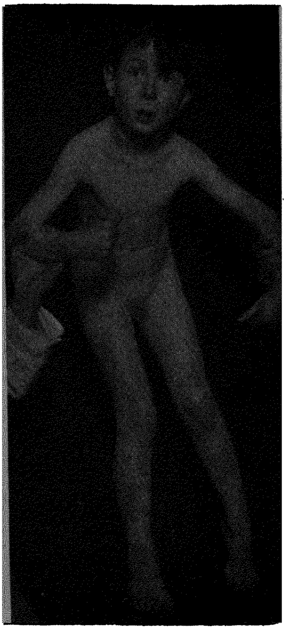

FIG. 92. Boy aged seven years, showing hysterical position of the left leg.

(1) A girl of ten years was brought because she was unable to raise her right arm or use her right hand. This had been so since an attack of chorea five months previously. During examination she was found to be able to grasp strongly with the right hand. After the application of mild Faradism to the right arm for a minute or two she was able to perform every movement quite normally.

(2) A little girl aged five years had attacks every few minutes in which she held her right arm in front of her face, and often managed to knock something over. When the right arm was tied to her side, the left arm took on the movements.

(3) A girl aged twelve years was brought because of her inability to sit up or walk since an attack of measles nine weeks previously. In hospital the night nurse reported that the child often sat up in her sleep. She was an odd child, frequently going off into hysterical giggles during the day, but under supervision she was soon able to sit up and walk and run.

(4) A girl aged ten and a half years was brought because of her unusual gait. After every few steps she would suddenly flex her left hip, raising the left foot high off the ground and swaying as if about to fall, which, however, she always just avoided. She was a restless, excitable child, and when spoken to would twist her body, clasp her hands and draw up her legs. She rapidly recovered on a course of bromide and valerian.

(5) A boy aged nine years had for three weeks been unable to walk or bend his back. There was no tenderness of the spine, nor evidence of organic disease. He made no progress in hospital until it was suggested that recovery would lead to his discharge home ; whereupon recovery was immediate.

Another form of motor hysteria sometimes seen in childhood is contracture of a joint, usually the knee, which may be so acutely flexed that the sole of the foot presses against the buttock, or may be rigidly extended. Any attempt to move the joint causes pain, and the contracture is best relieved under an anæsthetic. The limb should then be fixed in a splint or plaster for two or three days, to prevent any recurrence. Hysterical flexion of the hip, or spasm of the elbow or fingers, may also occur.

Hysterical convulsions are less common in children than in adults. They are to be distinguished from epileptic fits in that consciousness is not lost, the sphincters are not relaxed, the tongue is not bitten, the reflexes are normal, the colour remains good, and close observation often leaves no doubt that the child is out to attract attention. Less often sensory symptoms such as anæsthesia of glove and stocking distribution arise. Other manifestations are hysterical crying and screaming, attacks of mutism, and tight closure of one or both eyes.

Hysterical vomiting is also a rarity in childhood. The following case suggests that it may sometimes be founded upon some organic condition. A girl, eleven years old, had suffered for eight months from vomiting, either during or immediately after meals. X-ray examination showed evidence of cardiospasm with dilatation of the œsophagus. She was a thin, unhappy child, and in hospital was treated by the passage daily of mercury-filled œsophageal bougies—a treatment which she strongly disliked—and her weight remained stationary. It was then noticed that her palate was completely anæsthetic, and so the treatment was stopped, the bougies were discarded, and she was made to sniff a little salt and water up her nose before meals, being told that this would stop her vomiting and that she could then go home. Her vomiting stopped at once, and in the next six weeks she gained 25 lbs. in weight. An X-ray examination at the end of that time showed no change in the dilatation of the œsophagus.

Diagnosis. The diagnosis of hysteria is one that must always be made with caution, and never until a most thorough search for organic disease has been undertaken. Hysterical manifestations in children generally present some bizarre or contradictory feature which distinguishes them from organic conditions—the cases just described illustrate this quite well. Although with the aid of suggestion recovery can as a rule be speedily brought about, relapses may occur or some fresh form of hysteria may appear at a later date.

Treatment. Except in the mildest cases, which can be quickly cured by some simple method of suggestion, it is generally wisest to admit these children to a hospital or nursing home, where they will be away from their home environment and will be under the care of skilled nurses. The handling of the child needs to be firm, but vigorous coercion or harshness is quite unnecessary. Suggestion as a means of treatment can be applied more easily to children than to adults, and massage, passive movements, and particularly a weak Faradic current are useful adjuncts to suggestion. The child should not be allowed to regard himself as an invalid, and drugs are seldom required, but small doses of valerian such as m. 5 of the ammoniated tincture, combined with bromides, are sometimes useful, largely as a means of suggestion. After recovery, a few weeks of convalescence at the seaside should precede the return home.

CHAPTER XXII

MENTAL DEFICIENCY

Mental deficiency may arise from many causes and may appear in varying degrees, ranging from the most severe types of idiocy up to children who, in many respects, would pass for normal, but at school are slow to learn and soon lag a class or two behind their fellows. The legal classification (Mental Deficiency Act, 1927) recognises as "idiots" those who, by reason of their mental deficiency, are unable to guard themselves against common physical dangers. "Imbeciles" are those who cannot manage themselves or their affairs, but may be capable of simple employment. "Feeble-minded" persons are those who need constant supervision, and in childhood are unable to benefit properly from instruction in ordinary schools. As a separate class are the "moral defectives," who, for the sake of the rest of the community, require special supervision and control.

Roughly two-thirds of the cases of mental defect fall into recognisable clinical groupings, such as the cretin, mongol, microcephalic, and so on. The remainder are grouped together under the title of primary amentia, a group which comprises all those cases in which there is no obvious reason for the backwardness, nor do the children bear any physical stigma which would warrant their being placed in a separate class. The group is heterogeneous, of pre-natal origin, and probably contains various types as yet not sorted out. All degrees of backwardness may be met with in this group, from mere dullness with nothing in the child's appearance to make one suspect retardation, to children who are obviously idiots, and it becomes at once clear that there can be no uniform prognosis applicable to the whole of this group, nor any one standard method of treatment. Each case must be judged on its own merits, and treatment advised accordingly.

In addition to primary amentia, the following types of backwardness may also be recognised :—

Mongol.
Cretin.
Microcephalic.
Hydrocephalic.
Due to birth-injury, usually associated with spastic diplegia.

Epileptic dementia.
Epiloia (tuberose sclerosis)
Post-encephalitic.
Syphilitic dementia.
Familial cerebral degenerations :—
  Amaurotic family idiocy.
  Cerebromacular degeneration.
  Schilder's disease.

Most of these types are described under their appropriate sections in other chapters, but the mongol and the microcephalic child will be dealt with here. Before doing so there are certain general aspects of mental defect to be considered.

Etiology. With the exception of mongolism, which affects boys more often than girls, the sexes are about equally involved. It has commonly been held that a neuropathic inheritance exerts a strong influence in promoting mental deficiency, and in an analysis [1] of over 2,000 mentally defective children resident in mental hospitals a history of mental weakness or of epilepsy among the relatives was found in nearly half the cases. But the analysis was based on cases sufficiently severe to be in mental institutions, and is not a fair sample of mental defect as met with in hospital or private practice. The only types of mental defect of undoubtedly hereditary origin are the familial cerebral degenerations, which account for less than 0·5 per cent. of mentally defective children. A neuropathic inheritance may also play a part in microcephaly, epilepsy, and primary amentia, but is unproven ; certainly the parents of these children are generally normal enough. The figures of the late Dr. John Thomson [2] show that these forms of dementia constitute only a third of the total number of backward children.

Just as congenital deformities of one sort and another occur in undue proportion in the first born of families, so, according to the analyses of Still,[3] does mental deficiency show a special predilection for the first born, which is greater than can be accounted for by the special liability of primæ gravidæ to a difficult or prolonged labour. An exception to this generalisation must be made for mongols, who tend to appear lower in the family tree.

Whether the age of the parents has any influence on the likelihood of their offspring being backward is difficult to assess. The mother's age at the time of birth of congenitally deformed children is over, rather than under, thirty years, and one's impression is that the parents of mentally defective children also tend to be in the second half of their reproductive period. The consanguinity of parents also exerts an adverse influence, especially if there is any hereditary neuropathic taint in the family.

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[1] Shuttleworth and Potts, " Mentally Deficient Children," 4th edit., 1916.
[2] McNeil, C., *Brit. Med. Jour.*, 1934, *i.*, 584.
[3] Still, G. F., *Lancet*, 1927, *ii.*, 795.

The influence of syphilis and alcoholism in the parents has given rise to diverse opinions, but in this country the incidence of these two conditions is on the wane, and so their significance in the etiology of mental defect is becoming less important. It is, of course, known that congenital syphilis can produce definite degenerate lesions in the child's brain, but it is very doubtful whether parental syphilis can exert any other direct influence on the child's mind. That alcohol has a definite effect on the higher centres of the nervous system is common knowledge, and it would be reasonable to expect alcoholism in the pregnant woman to have an injurious effect on the young nervous system of the fœtus, and it is likely that maternal alcoholism is more harmful than alcoholism in the father. The general health of the mother during her pregnancy is also a factor to be reckoned with. It is certainly common enough for the mother to attribute the condition of her offspring to some illness or shock or fright sustained during pregnancy. It is known that certain acute infections (smallpox, measles, etc.) may be transmitted through the placenta to the fœtus, and it is not improbable that these and other acute illnesses of the mother may damage the delicate nervous system of her unborn child.

Diagnosis. As a general rule mental deficiency is not likely to be suspected until several months after birth, and then only because the infant fails to behave as do normal babies of his age. An exception to this is the mongol, whose typical facies dates from birth, while the features of the cretin are often sufficiently developed to allow of the condition being recognised by the end of the third month. In other cases the question of backwardness will depend on comparing an infant's attainments with those normal for his age, and therefore it is necessary to know the various milestones which are generally looked for when estimating a child's progress. Here it must be remembered that Nature lays down no fixed rules, and many children will develop in advance of the average. Parents are sometimes apt to expect too much of their offspring, and may fear a degree of backwardness when actually there is yet time for the child to grow within the normal limits.

During the first month most infants prove their ability to see by following a bright light, but blindness is generally difficult to detect until about six months, because the mere fact that infants take no notice of objects does not necessarily indicate blindness ; they may be so mentally backward that objects have no interest or meaning to them. Continuous rolling movements of the eyes are often evidence of defective vision. Also during the first few weeks most infants will start at any sudden noise, and by the fourth month will turn their head towards a sound, but a complete disregard of sound may as likely be due to mental defect as to deafness.

With regard to motor functions, even from birth infants have quite a powerful grasp, and an absolute failure to make any attempt to hold on to things is certainly suggestive of mental deficiency. Incidentally, it may be noted how often mentally defective infants will clench their fists with their thumbs inturned inside the fingers. At three months of age the infant should be able to raise his head, and by six months should be able to balance the head steadily while the body is supported. The power to sit up unsupported should be acquired by nine months, although many infants can do this earlier. Little importance attaches to crawling, since this is only a temporary stage, and is often omitted by a forward child. Between twelve and eighteen months infants should learn to stand unaided and to walk a few steps, but backwardness in walking should not be admitted until the child is eighteen months old.

The age at which talking begins shows much variation. Some children use simple words like "mum" and "dad" at twelve months, while others do not begin their vocabulary until two years old. Failure to talk by two and a half years constitutes backwardness in talking, but this may be due to causes other than mental defect, such as deafness or aphasia. Many children use certain sounds of their own to denote certain objects, and later replace these names by the proper words, but in backward children this baby language may persist for several years.

Backwardness in development may be due to physical causes, although the mentality may be normal. For instance, a child with amyotonia congenita is always very late in sitting up, and delay in walking may be due to such conditions as congenital dislocation of the hip or paralysis. In general it may be said that when the physical health alone is at fault the time of sitting up and walking will be delayed, but talking is not affected and the child takes interest in his toys and surroundings. On the other hand, mentally defective children will show a retardation of all phases of their development. Of course, it may happen that backwardness is due to a combination of mental and physical handicaps, as in cases of spastic diplegia with mental defect resulting from an injury at birth.

At a first glance there may be nothing in the child's appearance to raise one's suspicions, in fact, some infants are so placid that their parents think them perfect. But this in itself may be suggestive, for the normal baby should at times have a lusty cry, while mentally defective infants may lie quietly all day, hardly making a sound. The history of such cases often shows that there was great difficulty in getting the baby to suck during the first week or two, although a healthy baby has the sucking reflex present from the first day. On the other hand, spells of monotonous and uncontrollable crying may also be attributable to mental defect, although at the time it is always difficult to be sure that some better reason for the crying has not been overlooked. The behaviour of the baby may be noticeably odd as early as the sixth month. During the examination he may lie in his mother's arms motionless and apathetic, staring vacantly and taking no notice of his surroundings.

In older children there may be nothing in the facies to indicate mental defect ; in others the vacant expression with drooping mouth, protruding tongue, and constant dribbling leave no doubt of the amentia. Toys may either excite no interest or may be rapidly destroyed with unfailing regularity. Sudden and unprovoked bursts of crying are common and may be associated with impulsive actions. Some of these children develop a habit of frequently repeating odd gestures. Thus one little boy aged four years would sit on the floor for hours at a time, alternately rubbing the carpet as though polishing it and then with his hand to his nose making three short trumpeting sounds before returning to the carpet. Another child would often spend half an hour sitting on the floor with her forehead resting on the carpet between her feet. Others will sit contentedly in front of a window waving their fingers across their eyes to catch the alternate lights and shades. Another trick in those who are late in walking is to shuffle themselves across the floor by sliding on their buttocks. Troublesome constipation is a common accompaniment of mental deficiency in babies as well as older children. Control of the sphincters is generally a late acquisition.

A history of convulsions is obtained in a large number of mentally defective children, and the fits may be either of the grand mal or petit mal type. One is often asked whether the fits have accounted for the mental defect or *vice versâ*, and it is generally impossible to give a precise answer. As a rule both the mental defect and the fits are dependent upon some underlying cerebral condition. Recurrent convulsions do, however, affect the prognosis adversely, for the mental state is likely to show progressive deterioration. Other stigmata of degeneration have been described, such as a high narrow palate and deformity of the ears, but they are inconstant and may occur for other reasons.

Prognosis. " What will become of the child ? " and " What chance is there of improvement ? " are among the first questions that the anxious parents ask. In some of the types of mental deficiency the children are so alike that a prognosis for that particular type can be given with some measure of assurance. For instance, microcephalic children are the most difficult of all to train because of their tendency to become dirty, spiteful and stubborn, and for most of them institutional treatment becomes necessary. On the other hand, the mongol is a very imitative child, fond of musical sounds and good tempered. With assiduous training these children can be taught some measure of reading and writing, and in later life may even be put to do simple supervised tasks. Some varieties of mental deficiency, such as epileptic dementia, show a progressive deterioration, while others such as syphilitic dementia and the familial cerebral degenerations lead to a fatal issue.

Of all varieties, the prognosis of primary amentia is the most difficult to assess. Each case must be judged on its merits. Almost all of them will ultimately learn to walk, and speech of a sort will develop, while others who are merely dull may be able to benefit from the education of an ordinary school. It is, however, generally misleading to hold out any promise of sudden improvement when the child reaches some particular age. It is often surprising and gratifying to see how much improvement can be wrought by persistent and patient training, and, at any rate in the early years, it is unwise to adopt too gloomy an attitude. It is generally better to emphasise to the parents how much the child's future can be influenced by their own efforts at home, and in this way to encourage them in their long and arduous task.

The outlook as regards life in mentally defective children is not so good as for normal children. Their natural powers of resistance to infections are less, and many die in their early years, particularly from disease of the respiratory tract. In later childhood and adolescence the mortality is also higher than that of the ordinary population.

Treatment. The most important aspect of treatment lies in diligent training at home. With the exception of the cretin, who responds to thyroid therapy, drugs play a very minor *rôle.* The parents of a backward child are easily made desperate at the slow progress of their offspring, and unless the doctor is sure of his ground he should be careful not to increase their despondency by acquiescing too readily in the hopelessness of the case. Although cure of mental deficiency is not to be expected, most cases will respond to some extent to assiduous training, and at times the improvement surpasses expectations.

It is most important that training should begin as early as possible. One of the most important lessons to be learned is cleanliness, and much patience must be expended in teaching the child proper control of the sphincters. He should be sat out regularly after meals, encouraging an association in his mind between the feel of the chamber and the emptying of the bladder or bowel. Some children will indicate their need of the chamber by making some particular sound, and it is essential that this sound should be recognised quickly and its meaning acted upon.

The time that a child takes to learn to use the limbs will obviously depend partly on the condition of the muscles. When the musculature is flabby and hypotonic, light massage is valuable, but electrical treatment is likely to worry the child and can seldom be employed. Exposures to artificial sunlight seldom have the slightest effect. In spastic cases massage is contra-indicated, as it will only increase the muscle tone. Passive movements of all the joints should be carried out every day.

As soon as the child can pull himself up, walking may be encouraged with the aid of a push chair, which must be heavy enough not to be turned over. The child must also be taught to clothe himself, and every day he should help to put on one of his garments, and later on he must be shown repeatedly how to do up buttons. Clean feeding and the proper use of spoons and forks must be taught. When once the child has been taught to perform any action for himself, it should never be done for him, for he must be encouraged to take a pride in doing things for himself. When the movements of the limbs are inco-ordinate and clumsy, much ingenuity can be spent in teaching steadiness. For instance, the child may be taught to walk between the rungs of a ladder, or the floor may be marked out in various colours and the child be made to walk only on one colour. Stringing coloured beads together encourages steady movements of the hands. It is important to keep these children occupied as much as possible, and for this purpose musical or brightly coloured toys are very useful. When training the child to talk, it is as well only to teach one word at a time. The first words should be those which can be associated with toys or picture books, such as 'pussy' or 'dolly'. It must be remembered that backward children tend to pronounce their words indistinctly, and therefore care should be taken to see that words spoken to them are enunciated clearly.

Should undesirable habits appear, such as rocking to and fro, spitting or masturbating, they must be checked at once, otherwise they soon become difficult to eradicate. It is just as important that the child should know when he causes displeasure as when he is doing right, but, except in the case of deliberate defiance, corporal punishment should never be given, as it will frighten or antagonise the child and make the training increasingly difficult.

When the child reaches six or seven years of age, the question of special training and institutional treatment will often have to be considered. Before this age the child is usually better off at home. The circumstances of the parents and the presence maybe of other normal children in the family must be borne in mind. The advantage of a special school is that the training is in the hands of those best qualified to help the child make the most of his abilities, and there need be no fear that his intelligence will be dimmed by association with other backward children. If the child is to be brought up at home, he will need the constant supervision of his mother or a special nurse, and unless this is forthcoming he is better off at a special school.

The use of drugs is very limited. The occurrence of fits calls for the use of sedatives such as luminal or the bromides, and these drugs are often of service in other cases where the instability of the nervous system shows itself by restlessness, emotional upsets and outbursts of temper, for by their quieting effect they often seem to make the training easier. Thyroid is often prescribed, but except in cretinism its value is very doubtful, and sometimes it makes management more difficult in that the children become more restless and emotional.

The Mongol

The similarity in the appearance of all mongols is so striking that when once the characteristic features have been learned the children can be recognised at a glance.

In the typical mongol the skull is small and with practically no occipital bulge, the bridge of the nose is broad, the epicanthic folds are exaggerated and the eyes slope upwards from the inner canthus causing a resemblance to the Mongolian races and giving the condition its name. There is often a slight internal strabismus, and mongol babies frequently have a habit of rolling their eyes upwards, exposing the white sclerotic below the cornea. The mouth is foreshortened, and as a consequence the tongue is often protruding, and after three or four years the papillæ of the tongue become enlarged and the surface becomes much fissured. This characteristic appearance has been attributed to the habit of tongue sucking, but the fissuring does not appear until about the third or fourth year, by which time many mongols have been taught to keep the tongue inside the mouth, although even so the fissures appear. Protrusion of the tongue also occurs in other types of mental defect, and is a characteristic of the cretin, but the tongue in these cases does not develop the peculiar coarse fissuring which is so typically seen in the mongol.

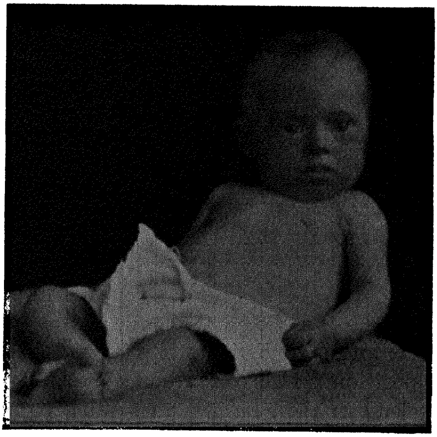

FIG. 93. Mongol baby aged seven months.

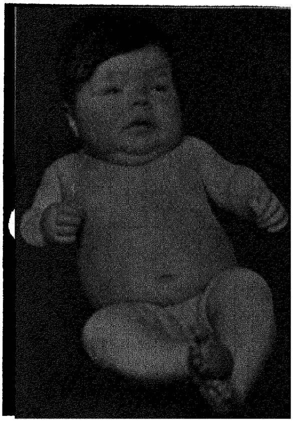

FIG. 94. Cretin, aged seven months. Compare with FIG. 90.

Snuffles is very commonly present, and there may be much purulent nasal discharge. The adenoid pad is enlarged, and soon becomes infected, and in addition the nasopharynx is so cramped for space that the airway is easily obstructed. When speech develops it is indistinct, and the voice is often coarse and rough. The hair is usually scanty, the ears are small, the hands are squat and the fingers are short and tapered, the little finger being often curved inwards. The creases on the sole of the foot are poorly developed, except for the deep crease between the big and the second toes. As a rule the muscles are flabby, and in consequence the limbs can often be put into contortionist attitudes. During infancy, mongols are generally plump in appearance, although actually their weight is likely to be below the normal, and as childhood advances the stunted growth becomes more and more obvious. After two or three years of age the skin is generally dry and coarse, and has a cyanotic tinge. Congenital heart disease occurs in about 10 per cent., and is an important factor in determining the high mortality during infancy.

Mental deficiency is invariable, and although it may reach a more severe grade in some than in others, the mental level does not rise above imbecility. Just as the physique of these children conforms to a definite pattern, so do their mentality and disposition. Mongols are, as a class, affectionate, but obstinate, clean in their habits, and quick to mimic the actions of others. They are happy so long as they are shielded from the teasing of other children. They always show a fondness for musical sounds and can often hum tunes from memory before they can talk.

Having pointed out how alike mongols are to each other, it must be remarked that degrees of mongolism occur as regards both mental and physical stigmata, but even if the traits are more conspicuous in some than in others, it is clear that these children have all been cast in the same mould.

There is nothing very characteristic in the post-mortem examination except that the brain is on the small side, the convolutions seem relatively few, and the cerebellar hemispheres are often so small that when the brain is turned upside down much more of the occipital lobes can be seen than is normally the case. Microscopical examination shows a general paucity of nerve cells.

The **diagnosis** of mongolism is an easy matter when once the typical appearance has been learned. The mongol is sometimes confused with the cretin, but this mistake should never arise. Cretins have a family likeness to one another and are recognisable at a glance, just as are mongols, but the two families have nothing in common. Some of the differences between mongols and cretins are set out in the following table :—

Mongol	*Cretin*
(1) Condition recognisable from birth.	(1) Condition not recognisable before the third month.
(2) Facies Mongolian, with eyes sloping upwards and outwards.	(2) Facies coarse, heavy lidded and eyes set horizontally.
(3) Complexion either pale or ruddy.	(3) Complexion butter-coloured.
(4) Expression often mobile.	(4) Expression dull.
(5) Tongue fissured, but scarcely enlarged.	(5) Tongue much hypertrophied.
(6) Hands small and fingers tapering.	(6) Hands broad, fingers thick.
(7) No supraclavicular pad of fat.	(7) Supraclavicular pad of fat.
(8) Abdomen plump.	(8) Abdomen protuberant, often with umbilical hernia.
(9) Constipation not a feature.	(9) Constipation profound.
(10) No appreciable response to thyroid treatment.	(10) Obvious response to thyroid treatment.

The cause of mongolism is unknown, but there are certain features in its etiology which have to be reckoned with in any satisfactory explanation. In the first place there is an amazing similarity in the physical appearance of all mongols, as though they were all members of one family. Another striking fact is their tendency to be born to parents who are reaching the end of their reproductive period. Thus the average age of the fathers of 200 mongol imbeciles was found to be 40·2 years, and the mothers of 196 mongols—37·2 years (Crookshank).[1] This has been expressed in a different way by Brousseau,[2] who showed that out of 457 fathers of mongols, no less than 85 per cent. were over 30 years of age (compared with a figure of 53 per cent. for the general population) ; while of 584 mothers of mongols, 76 per cent. were over 30 years of age (compared with 32 per cent. for the general population).

It would appear from these figures that if the age of the parents is a factor in the production of mongols, the responsibility lies no more with the mother—as has been suggested—than with the father. As one would expect from the figures given above, mongol children are often born towards the end of the family, but this is not necessarily so. They may be the first born, not merely of elderly parents, but also of young parents.

The customary explanation of mongols is that they represent an exhaustion product of a mother whose fertility is drawing to a close, but such an explanation is unsatisfactory for the following reasons :—

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[1] Crookshank, “ The Mongol in our Midst,” 3rd edit.

[2] Brousseau, Kate, “ Mongolism,” 1928.

(1) Mongols may be born to young mothers.

(2) It is exceptional for a woman to bear more than one mongol child. Should there be children born after the mongol, they will almost certainly be normal.

(3) When mongolism occurs in twins, almost always only one of the twins is affected. In those cases where both twins have been affected, they have either been uniovular or else the type of twins has not been ascertained.[1] Binovular mongol twins have yet to be described. If mongols were exhaustion products one would expect both of twins to be affected, but the evidence from twins seems rather to point to the condition being the result of some defect in the germ plasm. Crookshank has suggested on anthropological grounds that the mongol imbecile represents a reversion to an ancestral type.

Prognosis. The majority of mongols succumb during the first two years, which is accounted for in part by the high incidence of congenital morbus cordis in them, and in part by the low resistance which they show towards infections, particularly of the respiratory tract. Those who survive childhood do not get beyond the early years of adult life.

With regard to the mental condition, all stages of development are delayed, but walking and talking are eventually acquired, and some will learn to read and write. Owing to their happy and imitative disposition, these children are best trained at home during their early years, but later on they cannot be educated at an ordinary school, and either private tuition or else institutional training becomes necessary. Some of the less severe cases can be trained to such simple tasks as gardening or housework, but they will always require constant supervision.

Microcephalic Idiocy

Microcephalic idiocy means more than backwardness with a small head. The maximum circumference of the head at birth measures on an average 13 inches, and by the end of the first year this is increased to 18 inches. Measurements a little less than this are, however, common, and may not be accompanied by signs of mental deficiency. On the other hand, a head smaller than the average is common in other forms of mental deficiency besides true microcephaly. Brushfield and Wyatt found that out of 1,185 mentally defective children under eight years of age, 12 per cent. showed a cranial circumference of less than 17·5 inches, but only half of these showed the characteristics of microcephaly.

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[1] Rosanoff and Handy, *Amer. Jour. Dis. Child.*, 1934, **48**, 765.

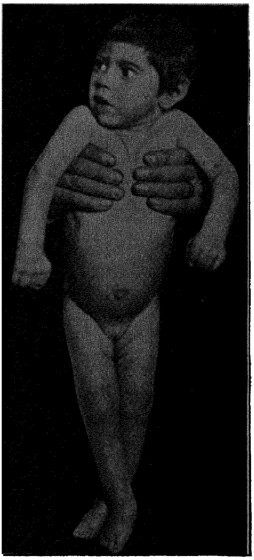

FIG. 95. Microcephaly. Note the spastic appearance of the limbs.

The head of the microcephalic idiot is always very undersized, and after two or three years may only measure 15 or 16 inches. The whole of the cranial vault is small, the forehead is very low and receding and the occiput is flattened, while the face, being of normal size, seems in striking contrast to the diminutive head. The eyes are set close together. The anterior fontanelle may be closed at birth, or closes soon after. As a rule the limbs are so rigid as to resemble spastic diplegia, although there is no actual paralysis. Epileptic seizures are a common complication.

The appearance of the brain varies. As a rule there is some thickening of the meninges, and the brain as a whole is small and conforms in shape to that of the skull. The hemispheres are particularly undeveloped. Although the brain is so small, the extent of cerebral tissue may in some cases be further diminished by internal hydrocephalus, or there may be areas replaced by cystic spaces (porencephaly). The varying appearance of the brain suggests that microcephaly may be due to several different causes operating during uterine life.

The degree of mental backwardness is roughly in proportion to the smallness of the head. Walking and talking are invariably delayed, and in the most severe cases the child may remain a bedridden idiot. Training of these children is always made difficult by their tendency as they grow up to become spiteful, dirty, and destructive, and eventually for most of them some form of permanent institutional management becomes necessary.

Moral Defectives

Occasionally one meets with children, usually in the second half of childhood, whose general mental and physical development has seemed normal enough, and who at school are quite as intelligent as their fellows, but who exhibit such freaks of behaviour as to make them appear devoid of any moral standards. Fortunately these cases are rare. Their abnormal behaviour may assume various forms. Some learn dirty bodily habits, or use obscene language, some are inveterate liars, others steal whenever the chance offers, and others are cruel and malicious towards other children or animals to a degree far exceeding ordinary mischievousness. Incendiarism is occasionally the predominant offence. No amount of punishment deters these children from their particular vice, and the proper handling of them is a problem which calls for the greatest care. As a rule there is some underlying cause for their odd behaviour, and much painstaking analysis may be required before the reason for their mental twist is brought to light. The exact history of the onset, and a knowledge of the environment at home and of the child's associates is most important. It may be that the child is allowed to feel unwanted at home, and he falls back upon his abnormal actions to secure for himself sufficient attention ; or he may have been spoiled until, with the arrival of a young brother or sister, he finds that he no longer holds the centre of the stage, and adopts an oddity of behaviour as a means of reasserting his position. In other cases the fault may date from some early impression of childhood. Some may undoubtedly be looked upon as congenitally defective, inasmuch as they have failed to develop that degree of cerebral attainment which is characterised by self-control and the power of inhibition.

The investigation and treatment of these children requires both time and patience, and is often best conducted at a Child Guidance Clinic. Not until the underlying factors have been exposed can any appeal to the child's reason be expected to be successful. If the cause can be remedied, all well and good, but if the factors are bound up with the environment, as, for instance, bad parental influence, it becomes necessary to remove the child to some suitable institution where training can proceed unmolested. In any case it is, as a rule, a good plan to get these children away for at least six months under the care of an intelligent and understanding foster-mother, who may be able to do much towards eradicating the trouble.

CHAPTER XXIII

DISEASES OF THE JOINTS, BONES, AND MUSCLES

On Aches and Pains in the Limbs—So-called Growing-Pains

ACHES and pains in the limbs are such a common complaint during childhood as to merit some comment. They do not of themselves, of course, constitute a disease, but are merely symptoms which may arise for various reasons. The term "growing pains" is unsatisfactory inasmuch as it implies that growth may actually be the cause of the pains, although growth is in fact a painless process. On the other hand, the term has the advantage of not implicating any particular disease, and it also lays emphasis on childhood as the period when these complaints are most common.

Children with aches and pains in their limbs are frequently regarded as rheumatic subjects, and there is a tendency at the present time to refer to them as suffering from "subacute rheumatism." This term has little to commend it. It is misleading if it is meant to imply a probability of more serious forms of rheumatism such as heart disease, rheumatic fever, or chorea, for only a negligible minority go on to exhibit at a later stage these more definite manifestations of juvenile rheumatism, and it is harmful in that it leads to many children being dubbed as rheumatic on the slightest grounds, and implants in them, and even more so in the minds of their parents, an unwarranted sense of invalidism. That the likelihood of serious rheumatic conditions befalling children with growing pains is remote was shown by following up 189 children who had attended the Rheumatism Clinic at Great Ormond Street for four years on account of aches and pains in the limbs ; during this period only two developed rheumatic fever with cardiac involvement, and none had chorea. The well-known histological changes which accompany rheumatic heart disease have never been shown to account for growing pains, nor is there the same relationship between sore throats and pains of this sort as exists between throat infections and subsequent acute carditis or rheumatic fever.

The question is generally approached from another angle—namely, that children with rheumatic heart disease often give a history of aches and pains in the limbs. A history of such pains was obtained in 52 out of 266 children with heart disease, although it does not follow that the pains were rheumatic. It is very noticeable that children with rheumatic carditis practically never complain of limb pains when they are away from home, as, for example, when at a convalescent home. Pains in the limbs are also common enough in other diseases; for instance, Bray [1] noted their occurrence in asthmatic children, and pointed out that there was no convincing evidence of their rheumatic origin. Similar pains may also occur in tuberculous children, and are common enough after the debilitating specific infections, especially measles and influenza. They frequently accompany chronic disorders of digestion, and are sometimes due to flat feet. Exceptionally they may occur as an early symptom of acute leukæmia, and are then due to leukæmic infiltrations beneath the periosteum.

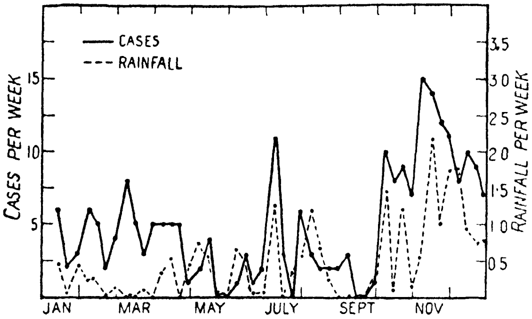

FIG. 96. Chart showing the incidence of pains in the limbs and rainfall during 1929.

Clinical Features. Growing pains are seldom met with under three years of age, and are most common between six and ten. The legs are more often involved than the arms, and the child can usually locate the pains in a rough sort of way, pointing to the region of joints or over muscle-bellies, but the joint surfaces are not affected—at all events movements of the joints are seldom restricted.

Generally speaking, growing pains fall into one of two categories, either they are related to changes in atmosphere, or else to fatigue.

Atmosphere. In about half the cases the pains bear a definite relation to changes in the weather, coming on particularly during wet weather. It is likely that dampness in the home or of the clothing contributes to these pains just as does wet weather. The close relationship between damp weather and growing pains is shown in the opposite chart, which gives the incidence of limb pains among children attending the Rheumatism Clinic at Great Ormond Street during 1929, and also the rainfall for that year in London.

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[1] Bray, "Recent Advances in Allergy," London, 1931, p. 393.

Not infrequently these children also complain of pains soon after getting into bed, or may be wakened at night by them, and it will then usually be found that they are sleeping between sheets. Pains which come on at night can often be abolished simply by putting the children to sleep between blankets.

Fatigue. When pains are due to fatigue they tend to come on towards the end of the day, often when the child gets in from school, and are relieved by rest. Children with these pains are often debilitated, and may show other evidence of physical and nervous exhaustion such as a sagging stance, postural scoliosis, and a functional albuminuria. In the same category are the leg pains complained of by obese children, and also the pains in the back of the legs associated with flat feet.

Different again are the muscular cramps, which occur particularly at night time, and may be very painful. The feet and toes are most often affected, and as a rule the peripheral circulation is poor, the feet being cold and clammy.

Treatment. The treatment varies with the type of pain. " Fatigue pains " call for adequate rest during the day and a course of general massage. Some such tonic as Parrish's Food may be given, and, if it can be arranged, a change of air to the seaside or country. Cramping pains are best relieved by vigorous rubbing. The children should sleep between blankets, and the bed should be warmed before they get into it. Pains associated with atmospheric changes are difficult to prevent. Sleeping between blankets should be advised, and during the daytime the warmth of the limbs should be safeguarded by long woollen stockings, gauntlet gloves, and stout shoes, and in wet weather provision should be made at school for the children to be able to change into dry shoes. The relief of the pains is best effected by rubbing the parts with some stimulating liniment. Liniment of methyl salicylate serves very well, although it is probable that the virtue of the liniment lies in the rubbing which goes with it rather than in its actual composition. Drugs, except as general tonics, are of little use. Sodium salicylate and aspirin, which are of great value in acute rheumatic arthritis, are disappointing when dealing with these more vague pains in the limbs.

Rheumatic Fever (Acute Rheumatic Arthritis)

This is a definite clinical entity characterised by acute non-suppurative inflammation of one or more joints, and must be clearly distinguished from the vague aches and pains in the limbs which have just been described under the title of growing pains. The two conditions have nothing in common.

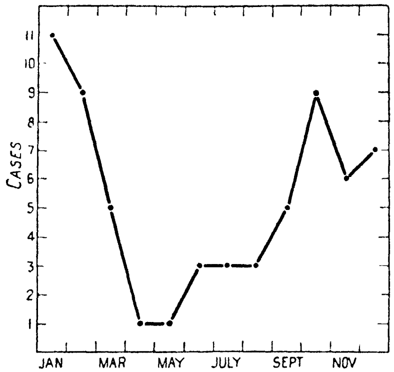


FIG. 97. Graph showing the monthly incidence of rheumatic fever (63 cases).

The claim of acute rheumatic arthritis to be included among the rheumatic phenomena of childhood lies in its frequent association with rheumatic heart disease, indeed it has been stated that every child with rheumatic arthritis may be assumed to have some carditis also, even if it be sometimes so slight as to give no recognisable clinical signs. Of 133 instances of rheumatic arthritis at the Rheumatism Clinic at Great Ormond Street, 93 (70 per cent.) showed evidence later of cardiac involvement, and in 5 instances the arthritis was followed by chorea.

Etiology. The remarks that have already been made in connection with the etiology of rheumatic carditis apply also to acute rheumatic arthritis, and therefore only the salient features will be touched on here.

Rheumatic fever is rarely encountered under three years of age. Of 133 instances the youngest occurred in a child of two and a half years old. The condition is particularly likely to occur in the cold wet months of the year, as may be seen from the accompanying graph. It has already been stated that an interval of from one to three weeks is likely to elapse between the onset of rheumatic heart disease and a preceding sore throat, and there is generally a similar time relationship between sore throats and rheumatic fever. The non-suppurative arthritis which is sometimes met with at an interval of a week or so after scarlet fever is identical with rheumatic fever, and is quite distinct from the suppurative arthritis which occasionally arises as a complication of scarlet fever.

Symptoms. The onset is brisk, with fever from 101° to 104° F.

and pain in the affected joints. The tongue quickly becomes coated with a thick fur, the urine is cloudy and high coloured, and anæmia rapidly develops. Sweating is sometimes a pronounced feature, but the sour and drenching sweats described in adults are not met with in children. Owing to the sweating, sudaminal rashes may appear on the trunk and limbs.

The affected joint is tender, and movements of it cause such extreme pain that the child may whimper with apprehension before the limb is touched. The other characteristic signs of inflammation, namely heat, swelling, and redness, are less obvious in children than in adults. It may be said that while pain is constantly present and the joint is usually hot, swelling only appears in about half the cases, and a red flush over the joint is but occasionally seen. The usual story is that fresh joints become involved every two or three days as those first affected subside, and in this way the illness may last for two or three weeks, unless checked by treatment. The joints most commonly affected are the knees, ankles, wrists, and elbows, in that order. Occasionally, and more often in children than adults, the inflammation is confined to one joint, which may lead to some difficulty in diagnosis.

Of the various complications the most important is rheumatic heart disease. It generally comes on at the same time as the arthritis, and is indicated by a rapid pulse, some degree of dilatation of the heart, shortness of the first sound, and perhaps a soft systolic bruit at the apex. Signs of pericarditis may also arise. Hyperpyrexia is an occasional complication, and is likely to be associated with much delirium.

Pathology. The affected joints are hyperæmic, and the synovial membrane is swollen. The exudate in the joints is turbid and albuminous, but not purulent, and is sterile on culture. Focal lesions comparable to the Aschoff nodules in the heart have been found in the synovia and joint capsules.

Diagnosis. As a rule the diagnosis is not difficult. The multiple acute suppurative arthritis of infants, which involves several successive joints as a result of pyæmic infection, is at once distinguished by the age, for rheumatic arthritis is not met with in infancy. Gonococcal arthritis may simulate rheumatism in that it affects several joints, but it may also involve the mandibular and sterno-clavicular joints, which are spared in rheumatism, and there is also likely to be the corroborative evidence of a vulvo-vaginal discharge. The immediately preceding history will serve to distinguish simple traumatic synovitis, while the more severe constitutional disturbance, the limitation to one joint, and the involvement of the neighbouring bones should prevent confusion with pyogenic arthritis or epiphysitis. Infantile scurvy and the syphilitic epiphysitis of infants belong to a much younger age group. One other condition deserves particular mention, namely, acute poliomyelitis, for the pain of rheumatic arthritis may be so sharp that the limb lies limp and motionless, simulating infantile paralysis, but a careful examination will show that there is no actual paralysis, the reflexes are not lost, and the tenderness is confined to the joints.

Special care is needed when only one joint is involved. If this should be the right hip, there may be enough rigidity over the right iliac fossa to suggest appendicitis. The big toe joint may also be picked out for solitary involvement, and in an older child might raise suspicions of gout, although gout is actually very rare in children.

Prognosis. As regards the arthritis the outlook is good, and complete recovery of function is the rule. The ultimate prognosis turns on whether the heart is involved or not, and the prognosis then becomes that of rheumatic heart disease. As with other manifestations of rheumatism, acute arthritis shows a tendency to recur, and a child may have as many as four or five separate attacks.

Treatment. The child should be nursed flat in bed between blankets, the weight of the bedclothes being held off by a cradle. Movements should be reduced to a minimum, and any necessary handling must be gently done.

So long as the temperature is raised the diet should consist chiefly of milk, milk puddings, fruit drinks, jellies and broths. Owing to the high fever and sweating, thirst is considerable and therefore simple fluids should be allowed in plenty.

The pain in the joints should be relieved by the local application of oil of wintergreen (lin. methyl salicylate). Strips of lint should be soaked in the oil and gently wrapped round the joint, being kept in position by several layers of cotton wool. Needless to say the liniment must not be applied by rubbing, owing to the extreme tenderness. Splints may be gently applied to keep the joints absolutely at rest.

Salicylates given by mouth have a specific effect in relieving the pain and reducing the fever, although whether they do anything to limit the occurrence of carditis is very doubtful, for the heart is usually involved at the same time as the joints.

(The employment of these drugs in the interval that elapses between a sore throat and subsequent arthritis or carditis is on a different footing, for they are then being used as a prophylactic before the rheumatic inflammation has developed.) In order to give relief quickly, the dose of salicylate must be adequate. 10 grs. of sodium salicylate should be given every four hours until the pain has been eased, and this dose should then be continued three times a day until the temperature has been normal for two or three weeks. If the drug is stopped too early the temperature may rise again and the joint pains may recur. It is most important that the dose of sodium salicylate should be prescribed with double the amount of sodium bicarbonate, in order to prevent symptoms of salicylate poisoning, such as vomiting, tachypnœa, and drowsiness Aspirin is as effective as sodium salicylate, and 10 grs. may be given three or four times a day, but being an acid substance it cannot be dispensed with sodium bicarbonate, and therefore if it is given over prolonged periods a careful watch must be kept for toxic symptoms. These are the same as those of sodium salicylate poisoning, the two most noticeable being vomiting and a hissing type of respiration. So reliable is the effect of salicylate in relieving the pain and reducing the fever that if after two or three days no response to the treatment appears the diagnosis will probably need revision.

There is sometimes difficulty in deciding when the child is to be allowed up. When the heart is involved, this will, of course, determine how long the child should stay in bed, but in the absence of physical signs of heart disease, convalescence should begin after a month. Convalescence should be unhurried, and a change of air to the country or seaside should be arranged before the return to school. During this period iron may usefully be given to overcome the anæmia.

Rheumatoid Arthritis

This condition is much less common in children than in adults, and the inflammatory changes are for the most part confined to the peri-articular tissues. Even in long-standing cases the actual joint surfaces may show little more than slight pitting of the cartilage, but the muscles gradually undergo extensive wasting from disuse, and eventually the bones in the affected areas become much rarified. In some children the changes in and around the joints make up almost the entire clinical picture, but more often there is an accompanying lymphatic reaction, as shown by enlargement of the superficial lymphatic glands and enlargement of the spleen (Still's disease).

In childhood the sexes are affected about equally, and there is no such predominance among females as is met with in adults. The age incidence is similar to that of acute rheumatism, inasmuch as rheumatoid arthritis rarely begins before three years of age. The average age of onset in eleven consecutive cases was six years.

The disease is usually regarded as infective in origin, the source of infection being perhaps in some such focus as the tonsils, teeth, sinuses, or bowel, but in many cases the origin of the infection is quite obscure ; nor has any causal organism been isolated. Instances occasionally arise which suggest that there may be some connection between rheumatoid arthritis and acute rheumatism, for example, there is often a history that the illness at its inception was regarded as rheumatic fever, but instead of recovering in the usual way the case slowly emerged as one of rheumatoid arthritis. More rarely there is coincident valvular disease of the heart, and Still has shown that at autopsy there may be diffuse pericardial adhesions and an adhesive mediastinitis, often unsuspected during life. In a few instances subcutaneous nodules of a persistent and fibrous nature have developed over the bony prominences.

Symptoms. The onset is often rapid, with fever and swelling of several joints ; in fact the illness may at first closely simulate rheumatic fever. In other cases the disease begins more gradually, and swelling and loss of movement of one or more joints may be the first complaint. Practically any joint in the body may be picked out, but the knees, wrists, elbows, fingers and ankles are those most commonly involved, and in that order. The cervical spine is often affected as well, causing stiffness and impaired movement of the neck.

As a rule the joints are affected symmetrically. They have a characteristic appearance ; owing to the swelling of the periarticular tissues they become fusiform in shape, and the skin over them may become shiny, while the degree of swelling is made even more obvious by the wasting of the surrounding muscles. On palpation they have a smooth elastic feel, which is particularly noticeable over the back of the wrist. The bony contour is often difficult to palpate owing to the amount of swelling of the soft parts, but osteophytic outgrowths do not develop. Pain is not a striking feature, but tenderness can generally be elicited on deep pressure. Invariably the range of movement is considerably limited, and in long-standing cases may be so extreme as to make the child bedridden. It is in such chronic cases as these that the hands gradually tend to deviate towards the ulnar side, the fingers become fixed in flexion, and the plantar arch becomes flattened out. Osteoporosis of the bones slowly increases until an X-ray film may show little but a rarefied shell of bone. Ultimately fibrous ankylosis is likely to come about, and at the wrist and ankle bony ankylosis may even take place, the X-ray showing a conglomerate mass of rarefied carpal or tarsal bones.

In most cases there is some enlargement of the superficial lymphatic glands, especially those draining the affected joints. Enlargement of the epitrochlear gland is so common and occurs so early as to be sometimes of help in making the diagnosis. The glands vary in size, but are usually as big as acorns, and are discrete, of a rubbery consistency, and not tender. Occasionally practically all the superficial glands are involved. The spleen becomes palpable in about half the cases, reaching from one to three fingersbreadth below the costal margin.

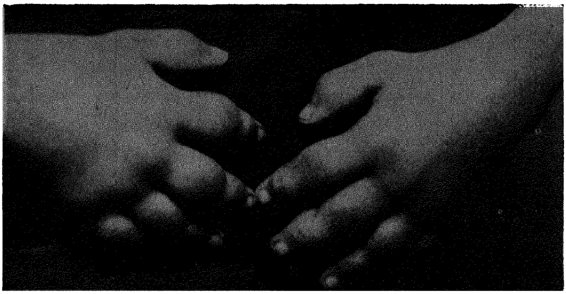


FIG. 98. The hands of a boy aged four years, suffering from rheumatoid arthritis.

There is considerable interference with the general health. Anæmia is usually present, and in the later stages a fine brown pigmentation may appear in the skin. The temperature is generally raised one or two degrees, with occasional exacerbations to a higher level, when fresh joints are likely to be involved. Sweating is common, and the extremities usually feel cold and clammy. In long-drawn-out cases growth is interfered with and the children become puny and stunted. The sedimentation rate of the red cells is raised so long as the disease is active, and repeated estimations may be used as a guide to the value of any therapeutic measure.

Complications. The occasional occurrence of subcutaneous nodules, adhesive mediastinitis, and valvular disease of the heart have been mentioned. Amyloid disease was a complication in one case, and mention is made later on of the rare association of rheumatoid arthritis with scleroderma (see p. 642).

Diagnosis. This should present little difficulty. Even when the onset is brisk, the less acute character of the pain and the failure to respond to salicylates distinguish it from acute rheumatism. Exceptionally a multiple synovitis occurs as a manifestation of congenital syphilis, but there will also be other signs, including a positive Wassermann reaction, to indicate spirochætal infection. The stiffness of the neck and spine might suggest spinal caries, but the involvement of the joints of the limbs will prevent this error.

Prognosis. The disease generally lasts for several years, the course showing periods of improvement followed by relapses. The ultimate prognosis is, however, by no means hopeless, indeed some children eventually recover completely, while others lose all trace of active infection but are left with permanently stiff joints. A recent enquiry by Colver into the after-history of 49 cases from the Hospital for Sick Children, Great Ormond Street, showed a mortality rate of 24 per cent. In each of the fatal cases the illness had started before five years of age, and death had occurred within three years of the onset. A fatal outcome is generally due to some intercurrent pulmonary infection.

Treatment. The first essential is to search for and remove any focus of sepsis. The writer has on more than one occasion seen the temperature fall to normal and a steady improvement take place after the dissection of septic tonsils. Abscesses arising from the roots of the teeth, and infected antra, are other sources of infection which may be easily overlooked. Whether intestinal toxæmia is really a factor is uncertain, but a regular action of the bowels is very desirable, and the diet should be as nutritious as possible. So long as the temperature is raised, rest in bed, preferably on a balcony, is advisable. Later on residence in a dry sunny atmosphere should, if possible, be arranged.

The assistance of a skilled masseuse is invaluable. Active as well as passive movements should be performed under supervision to preserve the mobility of the joints, and light splints may be required at night to prevent contractures and deformities. Local measures to the joints such as radiant heat, and heat given by applications of paraffin wax will help to relieve pain. Courses of ultra-violet light are also beneficial so long as the temperature is not raised more than a degree or two.

There is no specific drug. Salicylates seem to be entirely without effect, and more benefit is likely to be obtained from the prolonged administration of some general tonic such as equal parts of malt and syrup of the iodide of iron.

Intramuscular injections of gold salts are sometimes very beneficial, but during their administration a close watch must be kept for signs of toxicity. They should be reserved for those children showing fever, and in whom the sedimentation rate is raised. An initial injection of 10 mgm. should be given, and if no local or general reaction follows, the dose should be increased to 20, 50, and 100 mgm. Injections should be given once a week, and a single dose should never exceed 100 mgm. It is generally necessary to give two courses, with an interval of two months between them, the first course consisting of a total of ½ gm., and the second of 1 gm. Toxic manifestations include albuminuria, anæmia, and dermatitis, and during treatment the urine should be examined daily for albumen, and the hæmoglobin and sedimentation rate should be estimated each week.

Benefit has sometimes followed repeated induction of high fever (protein shock) by intravenous injections of foreign proteins such as T.A.B. vaccine, sterile milk, or peptone.

DISEASES OF BONES

Achondroplasia (Chondrodystrophia Fœtalis)

This remarkable condition is characterised by shortness of the limbs, leading to dwarfing, while the trunk remains of normal size. The disease may affect successive generations. The older name of " fœtal rickets," which was at one time applied to this condition, is misleading, for there is no connection between achondroplasia and rickets.

The long bones of the limbs are those chiefly affected. They are about half their normal length, but the shaft is stout and the ridges and markings are more pronounced than usual. At their ends the long bones are expanded and cup-shaped, at first sight being somewhat similar to rickets, and the epiphyseal centres are situated closer to the shaft than normal.

Clinical Picture. The appearance is so characteristic that the condition is recognisable at a glance. The outstanding feature is the dwarfing due to the shortness of the limbs, but this is only seen when the child is standing, for the trunk is not affected. There is generally some bowing of the limbs. The buttocks are

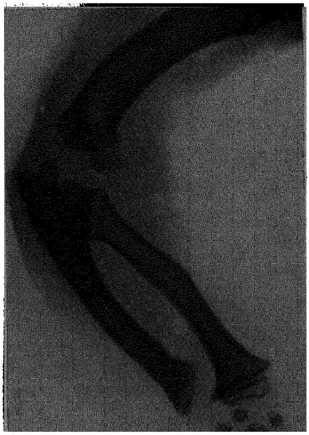
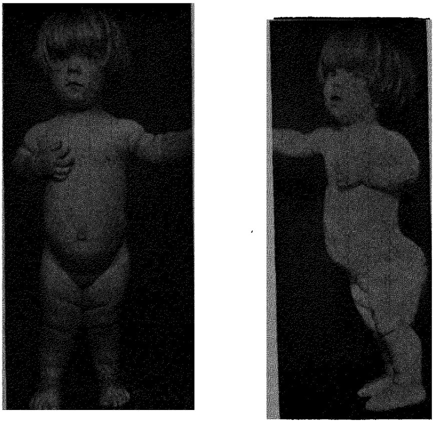


FIG. 99. X-ray appearance of the arm of a child with achondroplasia.

prominent, there is pronounced lumbar lordosis, and the child walks with a peculiar rolling or waddling gait. The arms are also so shortened that the hands scarcely reach to the hips, and the ends of the radius and ulna are bossed. The fingers are short and stumpy, and when outstretched give the appearance of a trident, the ring and little fingers being deviated to the ulnar side, while the middle and forefingers are deflected towards the radial side. There is usually some beading of the ribs.

The head appears large, and by measurement is actually increased above the normal, so much so that in young children the appearance of hydrocephalus may be simulated, and the features are sometimes rather coarse owing to the thick lips and the retroussé nose. The mentality is normal. Puberty occurs at the usual time and sexual activity is unaffected.



FIGS. 100 and 101. Achondroplasia.

Differential Diagnosis. The two conditions which are most likely to be confused with achondroplasia are rickets and cretinism. The disproportion of the limbs is, however, quite unlike rickets, and the X-ray picture of the two conditions is also quite distinct. Needless to say, the condition is uninfluenced by antirachitic treatment. The same points serve to rule out cretinism, in fact it is only the facial appearance which might at first cause difficulty.

Achondroplasia does not tend to shorten life, but is completely unaffected by treatment. Thyroid extract has been recommended, but usually has no effect.

Morquio's Disease

This rare condition was first described by Morquio [1] in 1929. From the close similarity of the recorded cases there can be no doubt that the disease forms a distinct clinical entity.

Several members of a family may be affected. Their appearance is striking, for they are dwarfed, the sternum is pinched forwards, and there is a dorso-lumbar kyphosis. Genu valgum is present, and the ends of the long bones are enlarged. The facial appearance and intelligence are unaffected. Radiologically the epiphyses of the long bones are grossly irregular. They are expanded, cup-shaped, and may show a fragmented outline, and the acetabula appear eroded. The vertebræ are wider and more shallow than normal, and at the level of the kyphosis one or more vertebræ appear wedge shaped, tapering anteriorly (Plate XI.).

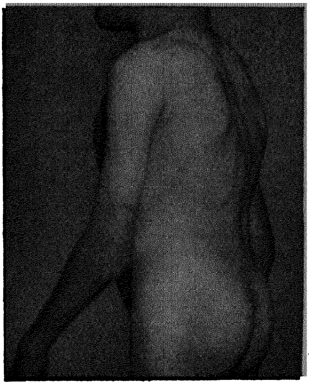


FIG. 102. Morquio's disease in a boy aged eight years. Note the depth of the chest and the dorso-lumbar kyphosis.

Gargoylism [2]

This is also a rare condition which at first sight bears resemblances to Morquio's disease, but the skeletal changes are more widespread, and other tissues are involved as well.

The syndrome comprises : a large head with frontal bossing, coarse ugly features, a wide mouth and broad tongue, a squat nose, nasal discharge, tough and dry scalp hair, and opacity of both corneæ. Lumbar kyphosis is present, and there is some limitation of movement of the joints of the limbs. The liver and spleen are enlarged, and there is a severe grade of mental defect. Radiologically the sella turcica is much elongated, but not deepened, the long bones are stouter than normal and their ridges and markings are accentuated, the epiphyses are expanded, and may be set at an angle to the shaft, and coxa valga may be present. The phalanges are also broadened. The kyphosis is due to deformity of one or more vertebræ, which show a peculiar hook-like process in place of the anterior half of the body (Plate XI.).

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[1] Morquio, L., *Arch. de Méd. des Enfants*, 1929, **32**, 129.

[2] Gargoylism. Ellis, W. R. B., Sheldon, W., and Capon, N.B., *Quart. Jour. Med.*, 1936, **29** (New Series, Vol. 5), 119.

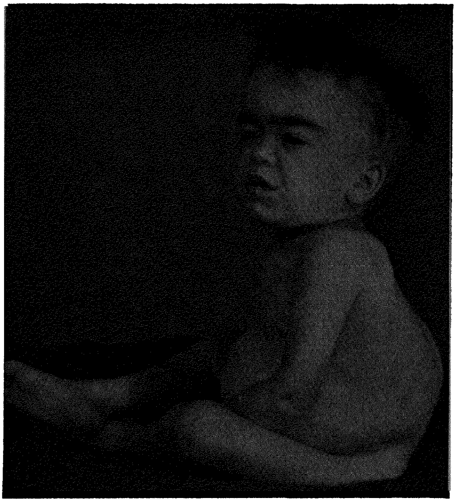


FIG. 103. Gargoylism in a boy aged three and a half years. Note the large head, coarse features, distended abdomen (due to enlargement of liver and spleen) and lumbar kyphosis.

Post-mortem examination has revealed that the changes in the soft tissues are due to the accumulation of a lipoid substance, which is at present unidentified. The parts predominantly affected by this change are the liver, spleen, anterior lobe of the pituitary, and the corneæ.

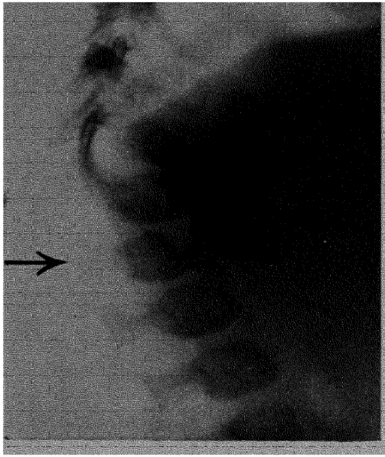
Both Morquio's disease and Gargoylism must be distinguished from the more severe degrees of infantile rickets, and from syphilis, cretinism and achondroplasia. The appearance of the hips in Morquio's disease must not be confused with pseudo-coxalgia (Perthe's disease), and this will be avoided if the examination is carried further than the hips.

Instances of both conditions have been recorded throughout childhood and beyond puberty. They are uninfluenced by treatment.

Osteogenesis Imperfecta (Fragilitas Ossium)

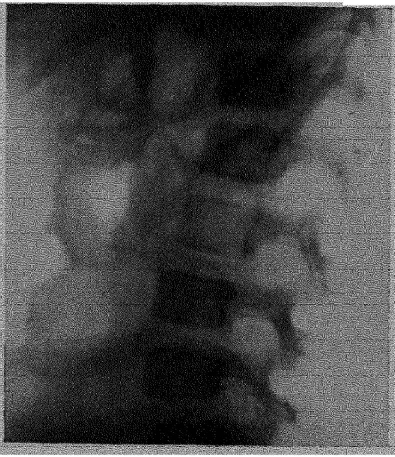
The outstanding feature of this remarkable condition is an undue fragility of the bones. There is sometimes evidence of fractures having occurred during intrauterine life, and multiple

PLATE XI.



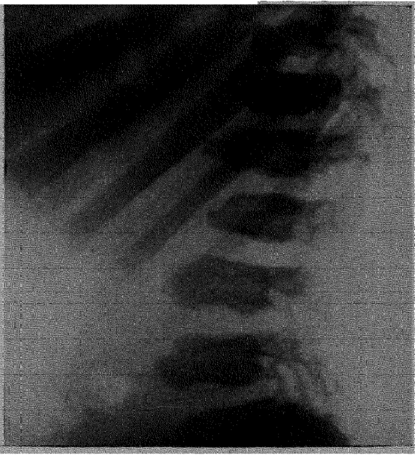
(A)

A. Gargoylism. To show the deformed second lumbar vertebra, indicated by the arrow.



(B)

B. Normal lumbar spine of a boy four years old. For comparison with figures A and C.

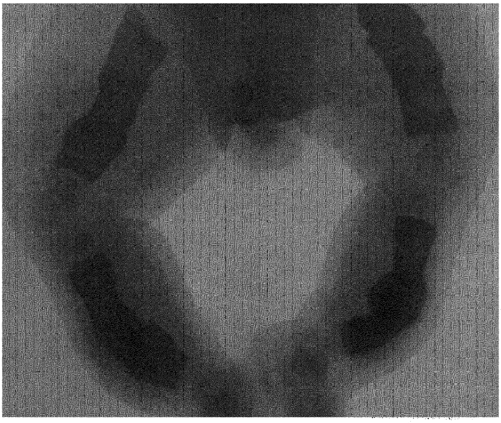


(C)

C. Morquio's disease. To show the narrowed and tapering lumbar vertebræ.

[*To face p.* 606.

PLATE XII.



Osteogenesis imperfecta. X-ray of the legs of the infant in Fig. 104.

fractures are likely to be produced during birth. In another group of cases the history of fractures does not begin until the third or fourth year, and in these children a peculiar blueness of the sclerotics is often noticeable. An hereditary and familial tendency is also more common in the latter group, but the difference between the two types is not sufficient to justify their consideration as separate entities ; more probably they represent varieties of one condition.

A familial history is obtained in about a quarter of the cases, and the condition has been known to affect both of twins. The sexes are equally involved. The changes in the bones vary somewhat with the age of the child. Soon after birth the long bones are broadened, stunted, rarefied, and much deformed in outline from the presence of numerous fractures and excessive callus (see Plate XII.), while in older children the bones appear slender, with a thinned cortex, and are often bowed. The fractures generally heal readily.

The cause is unknown. The formation of periosteal bone appears to be defective, and a reduction in the number of osteoblasts has been noted. Although the bones appear poorly calcified, the amount of calcium and phosphorus in the blood is normal.

Symptoms. The cases in which fractures arise at birth are the most severe. The bones are so soft that even the gentlest handling of the baby may produce a fracture, and as many as 100 fractures have been recorded in a newborn infant. The long bones become bent, and the attitude of the baby as he lies on his back with his arms spread limply away from the body, and the thighs and legs so bent as to make a circle with the feet together, is most striking. The vault of the skull is often so defectively ossified that in patches there is no bone at all, while in other places it is

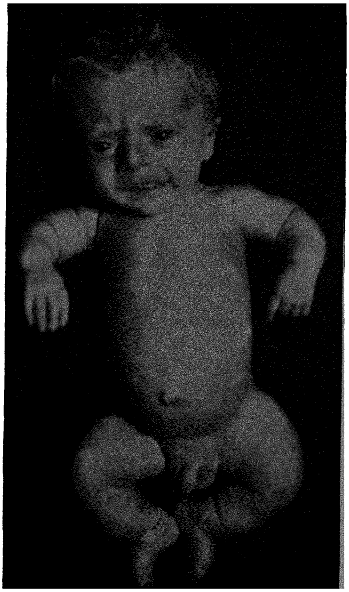


FIG. 104. Boy aged seven weeks, with osteogenesis imperfecta.

so thinned that craniotabes can be easily elicited. The majority of these infants die within a few weeks of birth.

In the group affecting older children fractures are easily produced by the mildest trauma, and not infrequently the break occurs at the site of a previous fracture. The bones gradually become deformed, and growth may be interfered with. The teeth are often soft, and may have a peculiar yellow translucent appearance owing to deficient calcification of the enamel. In these older children the outlook as regards life is good, although the deformities may greatly interfere with their activities.

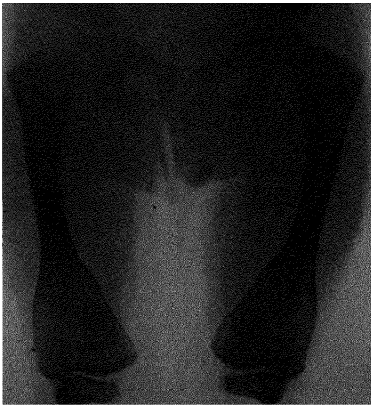


FIG. 105. The femora of a boy aged three years, with osteosclerosis fragilis. (By courtesy of Dr. Cockayne and Dr. Shires.)

Treatment. There is no curative treatment. In the infantile type the baby should lie on a bed of cotton wool and should only be lifted for changing. At all ages the greatest care must be taken to set the fractures in as perfect alignment as possible in order to reduce the likelihood of further deformity.

Osteosclerosis Fragilis (Albers-Schönberg Disease ; Marble Bones)

This rare condition was described by Albers-Schönberg in 1904. The long bones are much thickened towards their extremities, giving them a dumb-bell appearance, and are so densely calcified that on X-ray examination it is impossible to make out any trabecular structure, and the medullary cavity may be obliterated. In spite of the thickness of the bones they are peculiarly fragile, and fractures readily occur. Severe anæmia is often a prominent feature, and is due to a slow destruction of the marrow by the encroaching bone. Progressive loss of sight may be another distressing symptom, and is due to compression of the optic nerves as a result of narrowing of the optic foramina.

Instances in adults have been recorded, but the condition is essentially one of childhood, and apparently arises before birth. Both sexes are affected, and more than one member of a family may show the condition. Partial cases have been observed in which only one or two bones have been involved. The outlook is bad, and the majority of cases die during childhood.

Cleido-cranial Dysostosis

This rare condition is due to defective formation of the membrane bones, that is to say, the bones forming the vault of the skull and the clavicles. The condition may be both hereditary and familial.

The clavicles may be completely absent, but more often one or both ends are present while the remainder is represented by fibrous tissue. The muscles attached to the clavicle may be undeveloped. The effect of the deformity is to prevent the shoulders from being braced apart, and the child is able to approximate them so that they almost meet under the chin. The pubis may also fail to ossify.

Large patches of the skull may remain unossified, and the fontanelles may not close for several years. Bossing of the frontal and parietal eminences may appear. The mentality is unaffected, and the condition does not tend to shorten life.

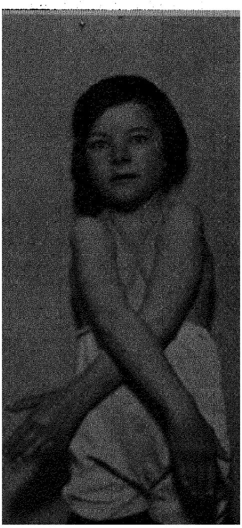


FIG. 106. Girl aged ten years, with cleido-cranial dysostosis, showing how the shoulders can be approximated.

There is no treatment.

Arachnodactyly

The peculiar feature of this rare condition is the unusual length of the fingers and toes, hence the name " spider fingers."

The long bones of the limbs may be affected also, but to a less extent. About half the cases show some form of congenital heart disease, and many show congenital ocular defects as well, such as dislocation of the lens, a rapid quivering of the irides, or nystagmus. There is a considerable degree of muscular weakness, which may lead to early death through intercurrent infections.

Congenital Elevation of the Scapula (Sprengel's Deformity)

Congenital elevation of the scapula may be bilateral, but more often only one side is affected, the scapula being raised an inch or more above its fellow. The scapula is also smaller than normal and tilted so that its lower angle lies close to the spine. In some cases it is fixed to the lower cervical vertebræ by tough fibrous bands or by a plaque of bone, and the arm cannot then be raised above the shoulder. Other deformities are likely to be present as well, such as scoliosis or torticollis.

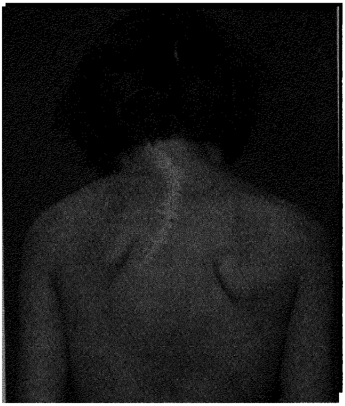


Fig. 107. Girl aged twelve years, with congenital elevation of the left scapula (Sprengel's deformity). A bridge of bone connecting the scapula with the spine has been divided.

As a rule treatment is unnecessary, but if a bridge of bone exists between the scapula and the spine its removal may lead to some improvement of function.

DEFORMITIES OF THE SKULL

Oxycephaly (Tower Head)

This deformity is due to a premature synostosis of the sutures at the base of the skull, and as the brain grows the capacity of the cranium has to be made good partly by an overgrowth of the great wings of the sphenoid, which bulges the skull laterally above the ears, but chiefly by an increased height of the vault. The forehead is abnormally high, towering upwards and forwards

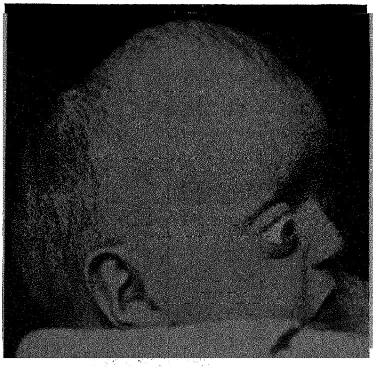


Fig. 108. Infant aged six months, with oxycephaly.

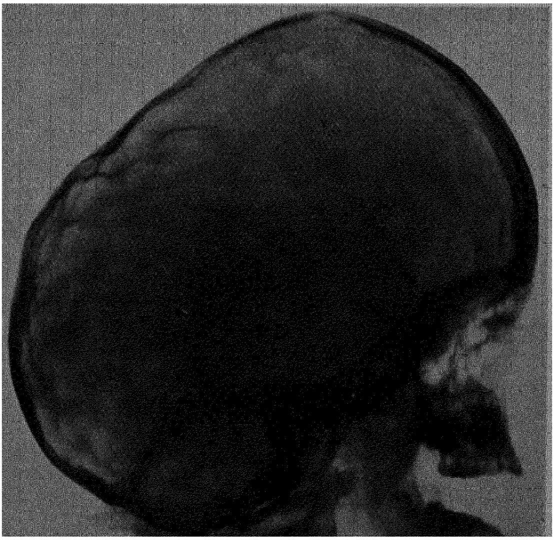


Fig. 109. Oxycephaly. X-ray of the skull of the child in Fig. 108, after he had reached six years of age. Note the digital markings.

over the brows, the highest point of the skull being at the anterior fontanelle. From this point the skull slopes backwards to the occiput. The orbits are wide apart and are so shallow that the eyes protrude, sometimes to such an extent that they are easily dislocated beyond the lids, and the optic nerves may be so stretched as to lead to atrophy and blindness. In spite of the remarkable appearance, the mentality is but little affected. As a rule only one member of a family is affected, but hereditary and familial instances have been recorded.

An X-ray of the skull shows characteristic digital markings over the vault, due to the pressure of the underlying brain.

Under the name " acrocephalo-syndactyly " a closely analogous condition has been described by Apert. In addition to a cephalic appearance resembling oxycephaly there is webbing of the fingers and toes, which may be so severely deformed as to be practically useless.

Treatment. In the face of advancing optic atrophy decompression may be necessary to preserve sight, but otherwise there is no treatment.

Scaphocephaly

This is a rare deformity associated with premature fusion of the sagittal suture and failure of development of the wings of the sphenoid. The skull is in consequence narrowed from side to side, but becomes elongated in an antero-posterior direction. Along the interfrontal suture the bone may be raised into a prominent ridge. As a rule the mental development is retarded.

Hypertelorism

This is another rare deformity of the skull first described by Greig,[1] and is the result of an overgrowth of the lesser wings of the sphenoid, combined with insufficient growth of the greater wings.

The facial appearance is characteristic. The bridge of the nose is very broad, and the eyes are set so far apart that the visual axes may diverge. The skull is also shorter than normal, the occiput is flattened, and there is pronounced lateral bulging in the temporal region. In spite of the thoroughly odd appearance, the mentality is usually unaffected.

There is no treatment.

Plagiocephaly

This is by no means an uncommon deformity, in which the long axis of the skull is slightly diagonal, one half of the frontal bone and the opposite occipital pole being unduly prominent, so that the vault of the skull has a twisted appearance. The condition is usually thought to be due to a malposition of the fœtal head *in utero*, and is sometimes associated with congenital torticollis.

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[1] Greig, D. M., *Edin. Med Jour.*, 1924, **31**, 560.

In the majority of cases the shape of the skull tends to right itself during the early years of childhood. No treatment is required.

SPINAL CURVATURE

Although curvature of the spine is not infrequently met with in childhood, the majority of the cases arise from surgical conditions and therefore will only receive passing mention here. The most important is tuberculous caries, and the physician needs to beware lest a history of pain, referred maybe to the front of the chest or abdomen, is in reality due to tuberculous disease of the spine. The pain may either be constant, or at first may only occur at night or when the spine is jarred, as in walking or going downstairs. Caries of the cervical spine must also be remembered as a possible cause of stiff-neck. Rigidity of the affected vertebræ when the spine is moved is a valuable and early sign, which may be detected long before the characteristic angular kyphosis appears. At a later stage the formation of abscesses may account for severe pains in the limbs, or paraplegia, or, if in connection with the cervical spine, may cause difficulty in swallowing or obstruct respiration.

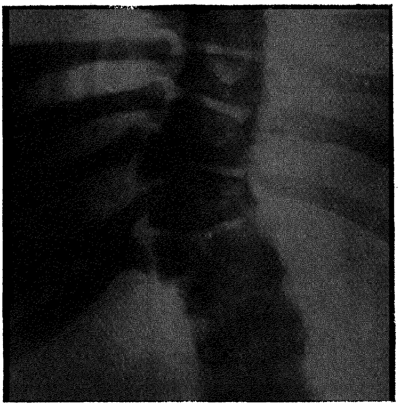

FIG. 110. Absence of the left halves of the tenth and twelfth dorsal vertebræ. There were only ten ribs on the left side.

Severe scoliosis is occasionally due to a failure of development of half a vertebra, and there is then generally a corresponding disproportion in the number of ribs. A scoliosis originating in this way appears as soon as the child begins to sit up. It is to be remembered also that the spine may become considerably curved to compensate for the tilt of the pelvis produced by such diseases of the hip as tuberculosis, congenital dislocation, coxa vara, or congenital shortening of one leg.

Atony or paralysis of the spinal muscles from such causes as rickets or poliomyelitis offer further causes of curvature.

Postural Curves. These are usually an exaggeration of the normal anatomical curves of the spine—in the lumbar region the normal lordosis is increased, while in the dorsal region scoliosis or kyphosis may appear, the latter being present when the child is said to be " round shouldered." Curves such as these are likely to accompany a general lowering of health from any cause, and are most common between about six and twelve years of age at a time when growth has been taking place rapidly. They indicate a state of physical exhaustion, and develop owing to laxity of the ligaments and loss of tone of the muscles concerned in posture, but are not due to actual disease of the vertebræ. The curves can usually be made to disappear by lifting the child off the ground. Functional albuminuria is a common accompaniment.

Treatment depends on improving the child's general health. To accomplish this, adequate rest, regulated exercises, and a generous diet are important. A change of air to the country or seaside is invaluable. A course of massage is desirable, and should be combined with special exercises to improve the tone of the muscles concerned in maintaining the upright posture.

Klippel-Feil Syndrome

This rare condition is the result of a congenital synostosis of several cervical vertebræ, which become fused into a solid bony mass. The neck is remarkably shortened, with a low hair line at the back, and lateral bending of the neck is limited. There may be an associated torticollis, facial asymmetry, nystagmus, and lesions of the brachial nerves.

DISEASES OF MUSCLES

Congenital Absence of Muscles

Congenital absence of part or the whole of a muscle is occasionally met with. The muscle most commonly affected is the costal portion of the pectoralis major. The defect gives a flattened appearance to the upper part of the chest, and is brought into prominence by making the child press his hands together. Not infrequently there is an accompanying deficiency of the portions of the ribs, namely, the second to the fifth, to which the muscle is normally attached. The function of the arm on the affected side is not noticeably impaired. No treatment is required.

Other muscles which sometimes fail to develop include the trapezius and serratus magnus.

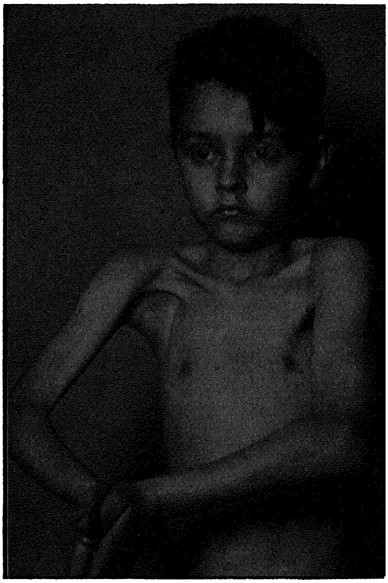

FIG. 111. Boy aged seven years, showing congenital deficiency of the right pectoralis major muscle.

Congenital Absence of the Abdominal muscles. In this condition the muscles and aponeuroses of the anterior abdominal wall are absent. The appearance of the skin of the abdomen is remarkable, for it is redundant and heavily creased. As would be expected, the abdominal contents are easily identified on palpation. The child is unable to cry lustily.

Almost always there is an associated deformity of the urinary tract. The bladder is hypertrophied, the ureters are dilated and tortuous, and there is a bilateral hydronephrosis. The failure of the abdominal muscles to develop has been attributed to pressure on them during intra-uterine life by the hypertrophied bladder, but it seems more likely that the deformity of the urinary apparatus is secondary to the muscle condition and is due to a lack of the normal intra-abdominal pressure.

The outlook is grave, and most cases die during infancy.

Amyoplasia Congenita[1]

(*Arthrogryposis multiplex congenita ; multiple congenital articular rigidity*)

This rare condition is characterised by rigidity of one or more joints of the limbs, generally with a symmetrical distribution. The fixation of the joints has the clinical appearance of fibrous ankylosis, and is associated with great diminution in size of those very muscles which would normally be concerned in producing the movements which the child is unable to perform. For instance, if the elbow is fixed in extension, the triceps will be found well developed, but the biceps is only a shadow of itself. The condition dates from intra-uterine life, and may possibly depend upon an arrested development of certain muscle groups. The joint surfaces and ligaments are only developed to an extent demanded by the range of movement.

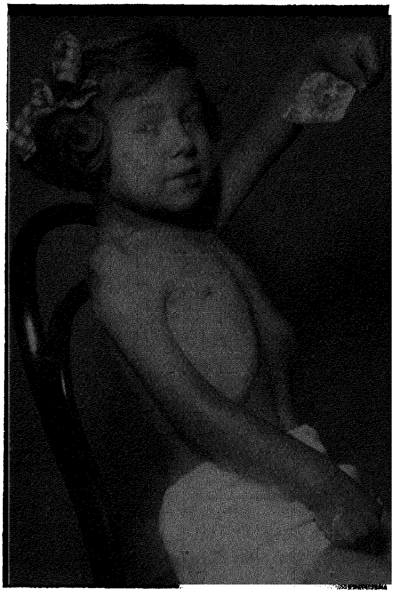

FIG. 112. Girl aged six years, with amyoplasia congenita. The elbows are fixed in extension. Note the wasting of the upper arm.

Clinically three types can be recognised :—

(i) All four limbs are fixed in a position of extension.

(ii) The hips are fixed in abduction, and the knees in flexion, so that the legs make a diamond pattern.

(iii) Occasionally the joints are affected asymmetrically, one side being fixed in flexion and the other in extension.

A slight amount of movement may come about after years of passive manipulations, while in others orthopædic operations may be undertaken to improve the alignment of the joints, but an increased range of movement is not likely to follow.

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[1] Sheldon. W., *Arch. Dis. Child.*, 1932, **7**, 117.

Torticollis (Wry-neck)

Wry-neck is a fairly common condition, and may be either congenital or acquired. Congenital wry-neck may appear within two or three weeks of birth in association with a sterno-mastoid tumour. Other cases are associated with a shortening and tightening of the sterno-mastoid muscle, and the appearance of the torticollis may then be delayed for a few months or even for three or four years, but once it appears it is persistent, and is likely to give rise to an asymmetrical development of the facies, the half of the face on the side of the wry-neck being smaller than the opposite half.

In cases associated with a sterno-mastoid tumour, daily passive movements should be carried out to preserve a full range of movement. The wry-neck passes off after a few months as the tumour in the muscle subsides. When the torticollis is persistent an open operation to divide the affected muscle is usually required.

Torticollis in older children is generally a temporary condition. It may be due to cervical adenitis or may be the result of exposure to draughts. The muscles concerned are tender, and efforts to straighten the head are painful. Relief is best afforded by the local application of heat, combined with massage.

Torticollis is occasionally due to ocular defects. The head is then tilted to one side, and the chin is also turned towards the same side. The position of the head can be easily and painlessly corrected.

MUSCULAR DYSTROPHIES

The muscular dystrophies are characterised by progressive weakness of certain groups of muscles, accompanied in all cases by wasting. In one type the weakness is associated at first with apparent hypertrophy of the muscles (pseudo-hypertrophic dystrophy), although later on these muscles also end in severe wasting. The muscular dystrophies affect successive generations, and several members of a family may show the condition, and when this is the case the disease is likely to appear at an increasingly younger age in the later members. Boys tend to be affected more frequently than girls. Pathological examination shows atrophy of individual muscle fibres and their replacement

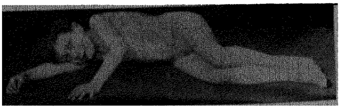


FIG. 113.

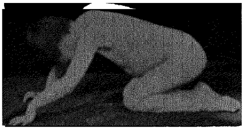
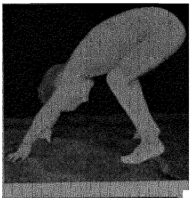


FIG. 114.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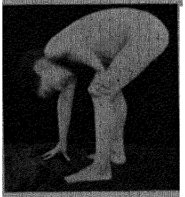
FIG. 116.

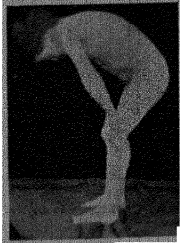
FIG. 117.

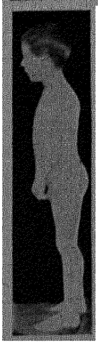
FIG. 118.

by fibrous tissue and fat. When fat replacement predominates, the muscles have the appearance of being hypertrophied and strong, although in reality they are weak, and as the fat slowly disappears wasting becomes evident. The nervous system is not primarily affected, although slight secondary degenerative changes may be found.

Three clinical groups are to be recognised.

Pseudo-hypertrophic Muscular Dystrophy. This is the most common type of myopathy. The symptoms first appear at about five or six years, although there may be a history of delay in sitting up and walking. When the condition is fully developed, the child waddles as he walks, rolling the body from side to side in order to clear his toes from the ground. Examination shows a mixture of hypertrophy of some muscles and wasting of others. The infraspinati and deltoids in the upper limbs and the glutei and gastrocnemii in the lower limbs are particularly hypertrophied and firm, and are in striking contrast to the wasting of the thigh muscles, the pectorales, the latissimus dorsi, and muscles of the upper arm. The face muscles and diaphragm generally escape. There is well-marked lumbar lordosis, which is thrown into relief by the large buttocks. The weakness of the shoulder girdle muscles is easily demonstrated by picking the child up under his arms, when, as in amyotonia congenita, the arms go up and the child tends to slip through one's hands. The method which

FIGS. 113–118. Pseudo-hypertrophic muscular dystrophy. A series of six pictures showing the characteristic manner in which a child rises from the prone position.

these children adopt in rising from a prone position is very characteristic. They first roll over on to the face, then draw up the legs under the body and straighten the arms so as to kneel with their hands on the floor; the legs are next straightened so that they stand on their hands and feet, and then they pull themselves up by working their hands up their legs.

There is no loss of sensation. The sphincters are unaffected, and the mentality is not impaired except in so far as the education of these children is likely to be neglected. The tendon reflexes gradually diminish and are finally lost.

The foregoing description represents the condition at the time when the diagnosis is usually made. During the second decade the hypertrophied muscles gradually waste, and eventually the patient becomes bedridden. Death usually comes about from intercurrent infection.

Facio-scapulo-humeral Type (Landouzy-Déjerine). This form of myopathy begins a little earlier than the pseudo-hypertrophic type. The facial muscles become atrophied, and the face is expressionless and devoid of wrinkles. The eyes cannot be properly closed and the lips protrude, giving a characteristic facies (*bouche de tapir*). The tongue, and muscles concerned in swallowing, are not as a rule affected, but the child is unable to blow a toy or to whistle. The muscles of the shoulder girdle also become weak and wasted, and at a later stage the pelvic and thigh muscles may become involved. The course is very slow, and the condition may remain stationary for several years.

Juvenile Type (Erb). This type begins towards puberty and affects principally the muscles of the shoulder girdle and upper arm, and the pelvic girdle and thighs. Winging of the scapulæ is a feature. There may be hypertrophy of certain muscles such as the deltoids and glutei. The course lasts over many years, and the condition may become permanently arrested.

Treatment. There is no curative treatment. It is important to encourage exercises as long as possible and not to let the patient take to his bed. Generally, splints and orthopædic operations are contra-indicated. The recent employment of glycine [1] has been reported to give some increase of strength to the muscles, without however leading to cure. The dose for children is 10 gm. daily, given orally in milk.

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[1] Cuthbertson, D. P., and Maclachlan, T. K. *Quart. Jour. Med.*, 1934, **27**, New Series 3, 411.

Myotonia Congenita (Thomsen's Disease)

This is a rare condition, showing both hereditary and familial tendencies. The characteristic feature consists of a curiously slow contraction and relaxation of muscles, although by repeatedly performing an action, as for instance shaking hands, it soon becomes more easily carried out. The symptoms may be noticed soon after birth, and persist into adult life. There is no evidence of involvement of the nervous system. The condition is painless, and does not tend to threaten life, but is unaffected by treatment. The stiffness must be distinguished from the rigidity of pyramidal paralysis ; in the latter condition the rigidity is not lessened by repetition of movements. Pathological examination shows hypertrophy and increased nucleation of the muscles.

Myasthenia Gravis

This condition, which is usually met with in adults, occasionally begins in the later years of childhood, and is dependent upon a delayed transmission of impulses at the neuro-muscular junctions. The chief symptom is an undue fatigue of muscles, the muscles of the face, the tongue, and those used in swallowing being principally affected, but the muscles of the trunk and limbs are occasionally involved. It is characteristic that the muscles recover after a short period of rest. The myasthenic reaction which obtains in this disease consists of the rapid exhaustion of the muscles by repeated Faradic stimulation.

The outlook is unfavourable. With careful treatment the patient may live several years and recovery occasionally takes place, but respiratory difficulties are likely, and death from exhaustion or intercurrent infection usually comes about within a year or two. The course is often marked by periods of remission.

Treatment. When swallowing is so difficult that there is a tendency to choke, food should be given through an œsophageal tube. Of drugs, ephedrine has been reported to lead to improvement, as has the administration by mouth of 10 gm. of glycine daily. Large doses of potassium chloride are also beneficial. The subcutaneous injection of prostigmin (an analogue of physostigmine) gives a dramatic but temporary return of power. The standard solution contains 0·5 mg. in 1 c.c., and an injection of 2 c.c. gives an improvement which lasts for about five hours. Larger or more concentrated doses are likely to cause toxic symptoms, particularly abdominal pain, which can be relieved to some extent with atropine.

CHAPTER XXIV

SOME COMMON DISEASES OF THE SKIN

Infantile Eczema

This may be defined as a catarrhal inflammation of the skin, of non-infective origin. Boys are more often affected than girls, and almost always the child is fair-skinned and blue-eyed, and above the average weight for the age. The appearance of fatness is, however, due to a watery distension of the tissues, and in the event of a sharp attack of diarrhœa the loss of fluid in the stools produces a rapid and severe loss of weight. The anæmic appearance of many eczematous infants is likewise due to œdema rather than to actual blood changes. The instability of the water-balance has been expressed in the term "hydrolabile," while the readiness with which eczematous infants develop catarrhal conditions such as bronchitis and diarrhœa led Czerny to suppose them to possess a special "exudative diathesis."

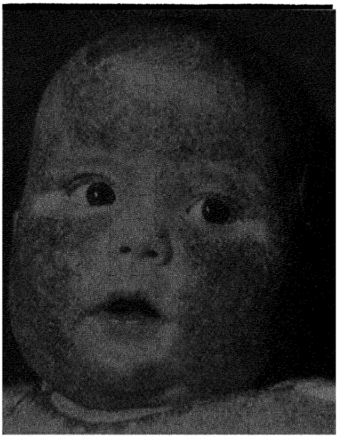

FIG. 119. Infantile eczema. Infant aged seven months.

Symptoms. The onset is seldom before the third month. The change from breast-feeding to a diet of cow's milk determines the onset in many cases, in others the rash first develops when the milk diet is augmented with other foods such as cereals and eggs.

The rash first appears, and is most intense, on the forehead and cheeks. At first the skin becomes red, and feels hot and dry, or may be thickened by œdema. Small papules and vesicles soon appear, and cause intense itching which the infant relieves by scratching until a thin serum exudes, or the skin may crack into minute fissures from which serum escapes. The serum dries into yellow crusts, and on the hairy scalp these may so coalesce that the head seems caked with them. Beneath the crusts the skin is bright red, raw, and moist.

The distribution of the rash may be either patchy or diffuse. Beginning on the forehead, the face and scalp are soon involved, and within a short time the trunk and limbs may be affected. The skin in the flexures and at the nape of the neck is often a favourite site. In older children there is not the same predilection for the face and scalp to be affected, more often the rash has a patchy distribution on the cheeks, chin, and limbs, especially in the flexures.

The skin is very liable to become secondarily infected, usually as a result of scratching, and the appearance of eczema is then masked by a septic dermatitis. This is particularly troublesome when it causes fissures behind the ears, the skin in these parts remaining affected long after the rest has healed.

Causation. There are two main views; some attach the chief importance to irritation of the skin by various external factors, others hold the sensitiveness of the skin to be due to various internal factors. Treatment is only likely to be successful when both views are taken into consideration.

External Factors. One of the most important factors in keeping up the rash is the constant scratching and rubbing. Other factors, such as sudden changes of temperature, exposure to winds or direct sunlight, washing with strong soaps and hard water, imperfect drying, and wearing rough or woolly clothing next to the skin, all help to aggravate the condition. It is probably the external factors which determine the distribution of the rash.

Internal Factors. The view that digestive upsets are the source of infantile eczema is no longer popular, although the rash is often worse if the infant is for any reason out-of-sorts or feverish, as for instance when teething and during vaccination. The fact that many eczematous infants exchange their skin condition for asthma as they grow older, and the frequent history of asthma or hay-fever in other members of the family, has led to the view that eczema is an allergic phenomenon, that is to say it derives from a hypersensitivity to foreign substances—usually proteins

—with which the infant comes in contact. The proteins of food, especially milk (lactalbumin), egg, and wheat, are the chief offenders, and about two-thirds of eczematous infants give positive skin-reactions to these or other proteins. Sensitivity is developed against the whole protein molecule, not against the products of protein digestion, and in this connection it is of interest that many of the infants show a deficient power of secreting hydrochloric acid (Bray), making it more likely that food-proteins may pass undigested through the stomach and be absorbed as whole protein from the intestine. The value of acid-milks in treatment probably depends on a more complete digestion of protein. Eczema is sometimes met with in breast-fed infants, and it has been shown that if the mother is achlorhydric, she may absorb the proteins of her diet and excrete them in her milk.

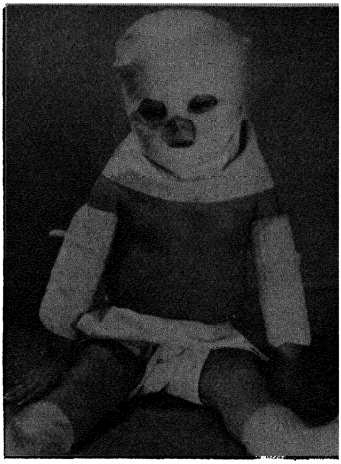

FIG. 120. To show the application of a face-mask and arm splints in infantile eczema.

Prognosis. Although the rash can sometimes be cleared up in a few weeks, to speak of " cure " would be incorrect, for unless therapeutic measures are continued—and indeed often in spite of them—the trouble is likely to break out afresh. Many children recover spontaneously towards the end of the second year, while in others, although a considerable improvement takes place, scattered patches continue to crop up throughout childhood. The replacement of the symptoms of eczema by those of asthma, both in infancy and older childhood, has already been mentioned.

The tendency to diarrhœa seems to account for those instances, fortunately rare, of sudden death in infantile eczema. The usual story is that the infant has done surprisingly well under treatment, but after a few days the temperature has mounted rapidly, severe diarrhœa has set in, and death has followed in a few hours. The following example serves as an illustration :—an infant aged six months had had generalised eczema since vaccination. After eleven days of treatment the skin became clear, but during the next night fourteen watery stools were passed, the temperature rose to over 105° F., and death occurred on the following morning.

Complications other than septic dermatitis are uncommon. A unique case was that of a male child six months of age who was admitted to hospital with severe eczema associated with pitting œdema of the legs and abdominal distension due to ascites. As the eczema improved under treatment, so the œdema and ascites quickly disappeared. The child remained under observation for four years, and although the eczema occasionally reappeared in patches, there was no recurrence of the ascites or the œdema.

Treatment. This falls under two headings—the protection of the skin from irritation, and internal treatment.

Protection of the Skin. It is essential to prevent the infant from scratching and rubbing the skin, and, except in the mildest cases, this can best be accomplished by applying a face-mask and arm-splints.[1] These should be worn until the rash has subsided. The infant should also be shielded from winds and direct sunlight, and the skin should be cleansed with olive oil instead of the usual soap and water bath. The clothing next to the skin needs to be smooth ; woollen and flannel garments are too irritating, and fine linen or cotton are best.

Before ointments are applied, any crusts should be removed. If bathing with warm olive oil does not get rid of them a starch and boracic poultice [2] should be used.

So long as the skin appears red and angry, a soothing ointment such as zinc cream should be liberally applied as a protective covering. If there is any superimposed infection of the skin, this should be dealt with at the same time by incorporating yellow oxide of mercury gr. 15 in the zinc cream.

When the acute inflammation has subsided, and in the more chronic forms with rough thickened skin, crude coal tar makes a most useful application, but its effect must be watched as it occasionally reddens the skin. It may be prescribed as crude coal tar 15 grains, zinc cream to 1 oz. Persistent patches of eczema on the face, chin, or limbs will sometimes respond satisfactorily to a combination of resorcin and birch tar in the following ointment: resorcin gr. 20, birch tar oil, m. 20, zinc oxide gr. 20, bismuth subnitrate gr. 20, hydrous wool fat, dr. 2, and soft paraffin to 1 oz.

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[1] The scalp and face are covered with a fine linen cap and mask in which holes are cut for the eyes, nose and mouth. Each arm is splinted by wrapping a strip of corrugated cardboard round the limb from the shoulder to the wrist, holding it in position by light bandages. The infant is able to wave his arms about, but flexion of the elbows is limited.

[2] *Starch and Boracic Poultice.* Mix a tablespoonful of starch with a teaspoonful of boracic acid. Stir to a stiff paste with cold water, then pour in boiling water until the starch becomes translucent. Allow to cool into a jelly. Apply by spreading the jelly on clean linen, and leave on for an hour. Repeat until all crusts have come away. This poultice may also be used in dealing with the crusts of impetigo contagiosa.

Internal Treatment. Drugs by mouth are of no value except in so far as the regular action of the bowels is concerned. When the skin is acutely inflamed ½ to 1 gr. of grey powder at night should be ordered. A low intake of fat in the diet is advantageous, and, if a dried milk is being given, a half-cream brand is preferable to one of full-cream strength. Of the two milk proteins, lactalbumin is the one to which eczematous infants are usually susceptible, and this may be removed from fresh milk by taking off the skin which forms after the milk has been boiled. The addition of hydrochloric acid [1] to the milk is also beneficial, because of the aid given to protein digestion. Freshly prepared acid milk has seemed to give better results than the various brands of dried acid milk. Almata, a synthetic milk-powder, is very successful in some cases, probably because it contains no lactalbumin, but both eggs and wheat are used in its manufacture, and if the infant happens to be sensitive to the proteins of these the rash may be aggravated.

When eczema arises in a breast-fed infant, it is generally unwise to advise weaning, for the trouble seldom lessens on cow's milk. Improvement may be brought about by giving the baby at each feed three drops of acid. hydrochlor. dil. (B.P.) in a teaspoonful of sweetened water. For the slightly older infant who has already started a mixed diet, the following mixture should be given in water in sips during meals, in which case the cow's milk need no longer be acidified, although removal of the lactalbumin (by taking away the skin formed after boiling) should continue.

℞ Pepsini gr. i.
Acidi hydrochlorici diluti m. viiss.
Syrupi limonis m. x.
Aquam ad ℥ i.

Ichthyosis

This is a congenital condition, although it may not be noticed until a few months after birth. The skin is dry, thickened, and scaly, the scales forming a mosaic-like pattern, and looking not unlike fish scales. They are often pigmented, making the skin appear dirty. The trunk and limbs are widely involved, but the palms of the hands, the soles of the feet, and the flexures escape, and the face is but slightly affected. A milder form of the condition, known as xeroderma, is not uncommon.

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[1] *Hydrochloric Acid Milk.* Boil 1 pint of fresh milk, allow to cool, and remove the skin. When cold, add slowly drop by drop forty-five minims of Ac. Hydrochlor. dil. (B.P.), stirring all the time. Only the finest curd should form. The milk may be given whole or diluted according to the infant's age and requirements. As with ordinary milk and water mixtures, the addition of sugar is necessary.

Like eczema, ichthyosis is often associated with asthma, but differs in that it is permanent, and shows no tendency to improve when the asthma develops. It may be added that asthma in these children is particularly resistant to treatment. Ichthyotic children are usually thin to the point of appearing badly nourished, and they feel the cold more than do normal children.

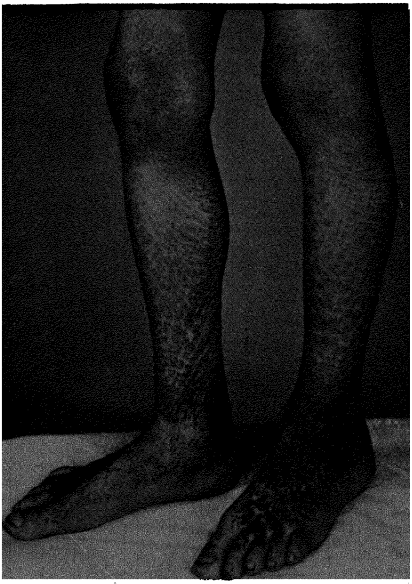

FIG. 121. Ichthyosis. Boy aged nine years.

Treatment. The condition is inborn, so that cure is out of the question. Thyroid gland is sometimes beneficial, and cod-liver oil and malt should also be given. Liberal inunctions of olive oil or vaseline may bring about some improvement, others have benefited from a daily bran-bath followed by local applications of glycerin and rose-water.

Psoriasis

This is seldom met with before the middle years of childhood. The lesions consist of circular dry patches, in size from a sixpence to a half-crown, of heaped-up silvery scales of epithelium on a dull red base. The patches can be scraped off with the nail, and leave a red erythema oozing one or two drops of blood. Acute cases occur in which the trunk, limbs, and scalp are thickly sown with patches, and under treatment these cases do well. In the more chronic cases the scalp, the front of the knees, and the back of the elbows, are the usual sites, and although under treatment these patches clear up, they are likely to reappear when treatment is stopped. The patches on the scalp may cause considerable itching.

Several members of a family may be affected. The cause is unknown. Some have supposed the condition to be a rheumatic manifestation, but there is no good evidence for this. Psoriasis does not affect the general health, and is not associated with any allergic state.

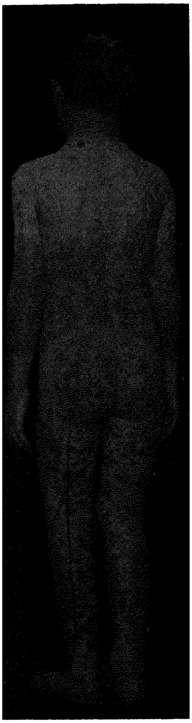

FIG. 122. Generalised psoriasis in a boy aged eight years.

Treatment. Reliance must be placed on local measures. The following ointment rubbed in night and morning gives satisfactory results :—

Liquoris picis carbonis ʒi.
Hydrargyri ammoniati gr. x.
Paraffini mollis ad ℥i.

Chrysarobin ointment (4 per cent.) has been recommended, but it stains the clothing, and may produce a severe erythema. If it is to be employed, its effect should first be tried on a small patch. The dithranol preparation *Cignolin* has properties similar to chrysarobin, but produces less staining of the clothes. Two grains may be added with advantage to each ounce of the ointment prescribed above.

Pityriasis Rosea

This is another eruption in which scaling is present, but its course is quite different from psoriasis or ichthyosis. The first lesion consists of a small pink scaling patch—the herald-patch—situated as a rule on some part of the trunk, and is followed in about ten days by numerous similar patches on the trunk and the proximal parts of the limbs. The patches are about the size of a threepenny-piece, and are flat and scaly, with a yellowish centre and a pale pink rim, and usually cause itching.

The rash disappears after six weeks or so, and does not recur. Treatment consists of a daily warm Condy's fluid bath, followed by inunction of salicylic acid (gr. 15 to the ounce of vaseline).

Lichen Urticatus (Papular Urticaria)

This is a variety of urticaria only met with in children, among whom it is very common, in fact most children suffer from it at some time or other in their early years. It is most frequent between six months and two years, but may continue up to about six years of age. It is more common during the spring and summer months.

The eruption begins as an urticarial wheal with a small red papule in the middle. The wheal fades after a few hours, by which time the papule has become vesicular, and, owing to the thick covering of epidermis, it has a hard shotty feel. The spots vary in number from a few to several hundred, and may occur on any part of the skin, but are most common on the buttocks and lower part of the trunk. On the soles of the feet and palms of the hands, where the skin is thicker, the vesicles may be ½ inch across, and are filled with a yellow opalescent fluid.

The eruption itches intensely, particularly when the child is warm in bed, and it is at night-time that the urticarial wheals are most noticeable. Scratching brings relief by rupturing the vesicles, leaving small pits in the skin which heal as a rule without a scar. A temporary brownish pigmentation sometimes marks the site of recent lesions. Needless to say, secondary infection may easily arise as a result of scratching.

More than one cause may be at work. Often there is some evidence of digestive disorder, the bowels may be constipated, or the stools may be unhealthy and contain undigested food particles and mucus, and on this account the rash has been attributed to the absorption of toxins from the bowel. An excess of cereals in the diet is sometimes responsible, and the condition is often present during the seasons when acid fruits are available. Bray has pointed out that bacon fat (including ham, lard, and bread fried in bacon fat) may be a cause.

Diagnosis. This is generally easy, but confusion may arise with chicken pox. The vesicles of varicella are, however, more delicate and thin-walled, they may be present inside the mouth, and a history of contact with another case may be obtained. Scabies is as great a cause of scratching, but a careful search of the skin will usually show the typical burrows of the acarus (see p. 643).

Prognosis. Although the eruption may quickly clear up under treatment, it is very likely to reappear, and it is extremely difficult to keep some children free from it. Even the most resistant cases grow out of the condition at about the time of the second dentition.

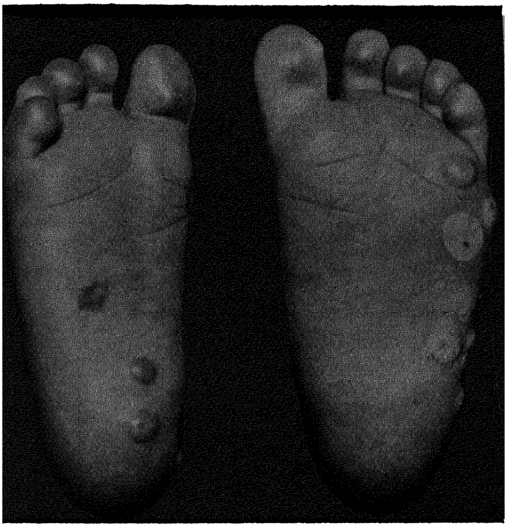

FIG. 123. Lichen urticatus showing the typical vesicles on the feet. Infant aged seven months.

Treatment. Local treatment is directed towards reducing the irritation and dealing with any secondary infection. A cooling lotion such as calamine lotion, or an alkaline lotion of sodium bicarbonate (half an ounce to 1 pint of water) should be applied frequently. Large blisters on the feet may be punctured and dusted with zinc powder. Secondary infections can be dealt with by an ointment of ammoniated mercury. The clothing needs to be light and smooth, cotton or fine linen being most suitable, and the daily bath should be cool, as the rash is usually worse after an ordinary hot bath.

Internal Treatment. The diet should be a light one, the last meal of the day being at teatime. Acid fruits (strawberries, raspberries, currants, gooseberries, plums, etc.), pork in any form, and breakfast cereals should be omitted. Sweets, chocolates, and those "nourishing" extras beloved by parents, such as cod-liver oil and malt and chocolate foods, should be avoided. A regular action of the bowels is essential, and may be obtained in young children by giving one or two grains of grey powder at bed-time, or the following mixture may be given after meals :

Pulveris rhei gr. i.
Sodii bicarbonatis gr. iv.
Syrupi zingiberis m. vi.
Aquam chloroformi ad ʒi.

In addition larger doses of alkali, such as half a teaspoonful of bicarbonate of soda in a small tumbler of water taken between meals, are of benefit.

Urticaria (Nettle-rash)

Urticaria is common to both children and adults. The rash appears as raised pinkish-white wheals or blotches on any part of the skin, and causes intense itching. The spots develop quickly and may only last an hour or so, or fresh wheals may continue to appear for several days.

The causes are various. Digestive upsets and constipation account for some, others follow rapidly after the ingestion of foods to which the child is sensitive, eggs, milk, porridge, fish, and fruits—especially strawberries and raspberries—being the most common offenders. Urticarial wheals are also caused by insect-bites and stinging nettles, and a severe urticaria is often a prominent symptom of serum sickness. *Dermographism*, in which wheals can be produced merely by lightly scratching or stroking the skin, is a rare form of the condition.

Treatment. Cooling applications, such as calamine lotion or zinc cream can be used to allay the irritation, and lukewarm alkaline baths may be given for the same purpose, allowing an ounce of sodium bicarbonate to 5 gallons of water. Salines such as milk of magnesia or magnesium sulphate should be given by mouth, and an alkaline mixture should be prescribed. Sodium bicarbonate, sodium citrate, potassium bicarbonate, and potassium citrate, 5 gr. of each in half a tumbler of water every four hours, does very well. A hypodermic injection of three to five minims of adrenalin may give temporary relief, and should be followed by a $\frac{1}{4}$ gr. tablet of ephedrine hydrochloride every six hours. A careful study of the diet in relation to the rash may indicate certain foods that must in future be avoided.

Napkin Rash (Jacquet's Erythema)

Napkin rashes are due in part to lack of ordinary care and attention in keeping the napkins changed and the parts clean, and partly to irritation and burning by chemical substances liberated from the stools or urine. Almost always the rash is associated with loose acid stools, sometimes caused by excessive amounts of carbohydrate in the diet, but more often due to the passage of fatty acids in the stool. The napkins often give off a strong smell of ammonia, the ammoniacal napkin burning the skin and accounting for the erythema. The ammonia is liberated in the napkin by the action of urea-splitting organisms from the unhealthy stools, which break down the urea of the urine into ammonia and CO_2.

At first the rash appears as a simple reddening of the skin, but if it is left untreated superficial blisters soon form, and rupture to leave extensive and often deep excoriations. The contact of urine or fæces against these raw areas is painful, and accounts for much screaming and restlessness. The distribution is as a rule sharply limited to the area covered by the napkin, and the rash is most severe at points where the napkin chafes, such as the convexity of the buttocks, the inner aspect of the thighs, the vulva, and the tip of the penis. If the infant is constantly drawing up his legs against the napkin the skin over the calves may also be involved.

Jacquet's erythema must be distinguished from the rash of congenital syphilis. As a rule the latter extends right to the anal margin, and may spread beyond the area of the napkin, and is a deeper red colour. There may also be other evidences of syphilis, such as a rash on the hands or feet, fissures round the mouth, snuffles, an enlarged liver and spleen, or epiphysitis.

Treatment. The liability to napkin rashes is, of course, much reduced as soon as the infant can be trained to pass urine or fæces into a receptacle, and therefore the regular " holding out " of the baby after each meal is an important preventive measure. Scrupulous attention must be paid to changing the napkins as soon as they become soiled, the parts being bathed with warm water and carefully dried and powdered.

If there is any soreness, the ordinary Turkish-towelling napkins should be lined with a thin inner napkin of soft material such as butter muslin, and strong soaps should never be used for washing the napkins. When the skin is reddened the napkins should be rinsed through a solution of perchloride of mercury (1 in 4,000) or boracic acid (1 in 100) after they have been washed, with the object of rendering them mildly antiseptic, and so inhibiting the growth of urea-splitting organisms. Each time the napkins are changed, the parts should be freely smeared with a protective grease such as vaseline, or if the skin has already broken, equal parts of zinc oxide and castor oil, or resorcin gr. 10 in vaseline ℥ i, should be applied. Attention must, of course, be paid to the correction of any dietetic errors.

Enema Rash

An erythematous rash occasionally appears about twelve to twenty-four hours after an enema. It is not due to any particular constituent of the enema as it may follow enemata of water, saline, or soap and water, but is the result of toxic absorption from the bowel. The rash may simulate that of scarlet fever, but is more usually a blotchy eruption occurring chiefly on the extensor surface of the limbs in the neighbourhood of joints and on the face, and it seldom lasts more than a day. It is not accompanied by fever or constitutional disturbance, is only slightly itching, and requires no treatment. Its importance lies in its recognition, for it must be distinguished from the rashes of the exanthemata.

Sweat Rashes

These are often seen in infants and young children, particularly in hot weather, or if there has been fever with much sweating. It will generally be found that woollen or flannel garments have been worn next to the skin. The rashes occur chiefly on the trunk, and may be either erythematous or more often consist of minute yellow sudaminæ, scarcely as large as a pin's head, on an erythematous base. The only treatment required is to lighten the clothing and to dust the skin with a simple powder such as equal parts of zinc oxide, boracic acid, and starch.

Drug Rashes

The common drugs that are likely to cause rashes in children are the bromides, belladonna, and phenazone.

Bromides. A bromide eruption is most often seen when the potassium salt is given over long periods, as, for instance, in the treatment of epilepsy, but occasionally a child is more susceptible and the rash then appears after only two or three doses.

The eruption consists of numerous small papules or boils, which do not irritate, and tend to dry off into scabs without leaving a scar. The face is most commonly affected. As soon as the drug is stopped the spots begin slowly to disappear, but may quickly reappear when the drug is restarted. It should be borne in mind that many soothing powders for infants contain bromide, and so may initiate a rash. The drug is also excreted in breast milk, and therefore a breast-fed infant may develop the eruption if the mother is receiving bromide.

Iodide rashes have a very similar appearance, but are not often seen in children.

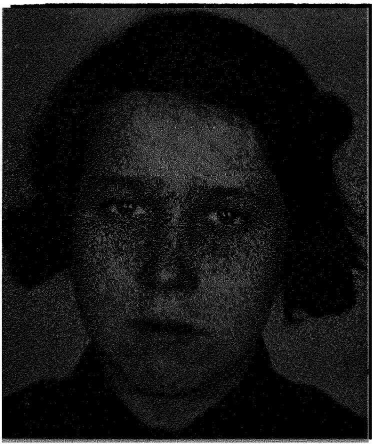

FIG. 124. Bromide eruption in a girl aged ten years, under treatment for epilepsy.

Belladonna. The dose required to produce a rash differs considerably with individuals, some children being able to tolerate a drachm of the tincture in the day without signs of intolerance, while others are affected by half or a quarter of this dose. . The rash consists of a bright red erythematous flush like that of scarlet fever, but is less widespread, being often confined to the neck and upper part of the trunk. Mental exhilaration, dilatation of the pupils, and a dry mouth are associated symptoms. The rash quickly passes off when the drug is withheld.

Phenazone (*Antipyrin*). This drug may give rise to a blotchy erythema on the trunk and limbs resembling the rash of measles, but the absence of fever, coryza, or Koplik's spots enables a distinction to be made. A similar eruption may be caused by nirvanol, a drug which has had temporary popularity in the treatment of chorea.

Impetigo

Impetigo occurs in two forms—impetigo contagiosa due to streptococcal infection, and Bockhart's impetigo due to the Staphylococcus aureus. The former is the more common.

Impetigo Contagiosa. As the name implies, this condition is highly contagious, and outbreaks of it may occur in schools, sometimes under the name "scrum-pox." The rash begins as small red papules which rapidly become converted into pustules, and then dry off into thick yellow crusts which are loosely attached to the underlying skin. The local lymphatic glands become inflamed, and may suppurate. The face is most commonly affected, and the crusts may be so clustered round the mouth and chin that little healthy skin can be seen. The infection may easily be transferred to other areas of skin either by direct contact or on the fingers. When the scalp is involved the probability of associated infection with pediculosis capitis must be remembered. Occasionally the rash spreads in rings—the circinate variety—which must be distinguished from ringworm.

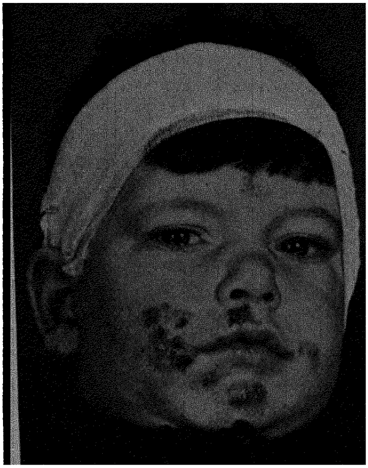

FIG. 125. Impetigo contagiosa, in a boy aged three and a half years.

Treatment should begin by the thorough removal of all scabs, which can best be accomplished with the aid of starch and boracic poultices (for preparation, see p. 624). A dilute ammoniated mercury ointment should then be applied freely, or if the infection is on the scalp a compress of perchloride of mercury (1 in 4,000) may be used. Fresh crusts must be removed morning and evening, as it is useless to apply ointment on top of them. Painting with 1 per cent. solution of gentian violet is an equally effective remedy, and is particularly useful for infected cracks behind the ears. Sulphanilamide powder freely applied is also successful. The child must also be prevented from rubbing or scratching the parts, and in young children a face-mask and arm splints should be worn (for method of application see p. 624). With efficient treatment most cases can be cleared up in a few days.

Bockhart's Impetigo. This consists of a staphylococcal infection of hair follicles, causing multiple pustules which vary in size from a pin's head up to an ordinary boil, and occur on any area of hair-bearing skin. Treatment consists of frequent bathing with hot water followed by compresses of perchloride of mercury (1 in 4,000). Ointments should not be used as they are likely to spread the infection. Boils should be fomented until they soften, and then should be opened and the boil and neighbouring skin be painted with weak iodine.

Both forms of impetigo are often benefited by exposing the skin to ultra-violet light.

Stye (Hordeoleum)

A stye consists of an abscess in the follicle of an eyelash. Styes are common in childhood, and sometimes recur on and off for several months. Treatment consists of warm bathing or fomentations, and the appropriate eyelash should be plucked. The child must also be discouraged from rubbing the eye, as this is likely to spread the infection. When a succession of styes occur a staphylococcal vaccine will sometimes bring them to an end. Cod-liver oil and tonics are also indicated.

Lupus

This is the result of infection of the skin by the tubercle bacillus. It is a slowly progressive condition which often starts in childhood, at which age it is most amenable to treatment.

The essential lesion is a lupus nodule, which at first is single, although by the time the child is brought for treatment a small patch of nodules has already formed, causing a dull red thickened and infiltrated area of skin. Pressure with a glass slide causes the erythema to fade, and then the nodules can be seen as light brown "apple-jelly" granules hardly larger than a pin's head. By pinching up the skin its infiltrated character can be felt. The first lesions are generally on the face, but any area of skin may be affected, and if left untreated the patches slowly spread. The mucous membrane of the mouth and nose may be involved, and the underlying cartilages may become eroded. Ulceration of the patches may occur, leading to considerable crusting.

Treatment. The local application of ultra-violet light often gives good results, but the special Finsen light treatment is the most satisfactory. It requires, however, special apparatus, and can only be carried out by those versed in its technique. Small patches may be treated by excision or by scraping with a sharp spoon. Alternatively caustics may be applied. Adamson [1] recommends either an ointment of pyrogallic acid (40 per cent. in vaseline) applied on lint for forty-eight hours to cause ulceration, after which the area is cleaned and fresh ointment is put on until the area is healed ; or the areas may be mopped with liquid acid nitrate of mercury until they become white and opaque. The painting is repeated every few weeks until no nodules remain.

The general health must also be improved by a generous diet, cod-liver oil, and open air.

Erythema Nodosum (Nodal Fever)

This remarkable condition is not uncommon in the second half of childhood and in adolescence, and is met with more often in girls than boys. As a rule the first evidence of the disease is the sudden development of the typical red areas on the shins, although further inquiry may show that the child has been vaguely ailing in health for a few weeks. There is often a history of a sore throat, or definite tonsillitis, about a fortnight previously. The development of the spots is accompanied by fever of two or three degrees, and sometimes by a sore throat and pains about the joints.

The eruption is so typical that it can hardly be confused with other conditions. Down the front of the shins there appear red raised indurated and slightly tender swellings, varying in diameter from ½ inch up to 2 or 3 inches, and in number from two or three up to a dozen or more. The nodules are either circular, or oval in shape with their long axis in the direction of the limb, and at first they are bright red, but in a day or so the centre turns a dusky colour. Each nodule lasts about a week, and as it subsides it turns purplish, finally leaving a brown staining. The nodules never ulcerate nor suppurate, and the regional lymphatic glands are not involved. As the first nodes disappear fresh ones may form, and so the condition may continue for two or three weeks. Occasionally nodules also appear above the knee and on the arms, but always they are more thickly grouped on the shins.

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[1] H. G. Adamson, "Diseases of Children," edited by Garrod Batten Thursfield and Paterson, 1929.

Various theories have been put forward in explanation of the condition. It has been looked upon as a specific disease due to an unknown virus, but there is little good evidence for this. It is true that small epidemics in families and dormitories have been described, but it does not follow that the disease is therefore communicable from one child to another, nor does one attack confer immunity. Originally erythema nodosum was held to be a rheumatic manifestation, but a history of it is very seldom obtained in children who have suffered from other serious forms of rheumatism such as carditis, arthritis, or chorea, nor do children with erythema nodosum show a predilection to develop these other rheumatic manifestations in later years.

Attention has recently been focussed on the frequent association between erythema nodosum and early tuberculous infection elsewhere in the body. In an inquiry into the after-history of 288 cases Ustvedt and Johannessen [1] found that within five years 25 per cent. had developed some form of tuberculosis. Many cases give positive skin reactions to tuberculin, while others show clinical and radiological evidence of enlarged mediastinal glands. A study by Wallgren [2] of an epidemic affecting 12 out of 34 scholars in a school class showed that all the patients gave positive tuberculin reactions, and half of them showed X-ray evidence of mediastinal adenitis ; one scholar was found to have active phthisis, and was thought to have accounted for a wide dissemination of tuberculosis among her schoolmates. In London, Collis [3] found that 70 per cent. of a group of children with erythema nodosum gave positive skin reactions to tuberculin, while others gave positive skin reactions to a similar preparation made from hæmolytic streptococci. The disease has also occurred in the course of diphtheria, cholera, and meningococcal infection. It seems clear from the foregoing that erythema nodosum may be associated with a variety of bacterial infections, usually tuberculous or streptococcal, and the most recent view of its pathogenesis is that it marks the development of a state of allergy or hypersensitivity to bacterial proteins liberated from some distant focus of infection in the body.

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[1] Ustvedt and Johannessen, *Norsk Magasin for Nægevidenskaben*, Oslo, 1933.

[2] Wallgren, A., *Jahr. f. Kinderh.*, Berlin, 1927, **117**, 313.

[3] Collis, W. R. F., *Quart. Jour. Med.*, 1932, New Series, Vol. **1**, p. 141.

Prognosis and Treatment. The prognosis is good as regards recovery, and complications are exceptional. A patient may, however, have more than one attack, and one child observed by the author experienced five attacks.

Treatment of the eruption is hardly called for beyond rest in bed, using a cradle to raise the bedclothes off the legs. Aspirin or salicylate of soda may be given to reduce the temperature and to relieve any tenderness.

The importance of erythema nodosum lies in the fact that it may be the first warning of recent tuberculous infection, and it therefore behoves the physician to satisfy himself on this point before discharging the patient. A careful physical examination together with X-ray evidence may lead to the discovery of an early focus in the chest, and further proof may be gained by recovering the tubercle bacillus from gastric washings. Other members of the family should also be examined, as one of them may be acting as an unsuspected source of infection.

Herpes Zoster

This condition, which is not uncommon in children, is characterised by a painful vesicular rash distributed in the area of skin supplied by one or more sensory nerves. For some hours before the spots appear the child may be feverish and complain of pain and tenderness over the part about to be affected. Small erythematous patches then appear, on which little blisters form, each about the size of a pea. The contents are at first clear, but turn yellow in a day or two, and then dry off into scabs which separate in a week's time without leaving a scar. Scarring may, of course, occur if the child scratches and causes secondary infection. The rash is usually distributed in the form of a half hoop two or three inches wide encircling precisely one half of the body, and corresponding to the distribution of one of the thoracic nerves. Less often an area of skin on a limb may be affected. *Herpes Frontalis* denotes involvement of the skin supplied by the supraorbital division of the fifth cranial nerve. Not only is herpes in this situation very painful, but the eye may also be involved, with conjunctivitis, keratitis or iritis. The face, mouth, or palate are other situations which may be affected.

The pathological changes consist of inflammation of one or more posterior root ganglia. The disease is caused by a filter-passing virus allied to, if not identical with, that of chicken pox. One attack does not confer immunity.

Treatment. The course of the disease cannot be shortened, and treatment simply consists of resting the child in a position which will relieve pressure on the part involved, and dusting the spots with zinc and starch powder. Sedatives such as bromide or chloral should be given so long as there is pain.

Herpes Febrilis. This bears no relation to herpes zoster. It consists of a small cluster of vesicles usually on the upper lip or at the corner of the mouth. The vesicles dry up into crusts which are shed without leaving scars. The condition is often seen as an accompaniment of a common cold, and it may also occur in pneumococcal pneumonia and meningococcal meningitis. Occasionally it is met with as a recurrent condition, herpetic vesicles, a raised temperature, and joint pains appearing at intervals of a month or so, each bout lasting up to a week, and the whole illness spreading over four to six months. The general health is but little affected. It is caused by a filter-passing virus closely related to the virus of encephalitis lethargica.

Treatment. This consists of protecting the parts with a bland ointment, such as cremor zinci.

Sclerema

This curious condition affects very young infants, and is characterised by a patchy, or more rarely diffuse, solidification of the subcutaneous fat. Other names that have been given to it include subcutaneous fat necrosis, subcutaneous fat sclerosis, scleredema, and pseudosclerema.

The condition picks on weak and puny infants, the symptoms appearing as a rule a few days after birth. Patches of the skin become lumpy, raised, and have a firm feeling like indiarubber. The skin is attached to the subcutaneous tissues so that it can be picked up, but the patches can be moved on the deeper structures, and they are not tender. Often the overlying skin has a bluish cyanotic tinge. The situations most likely to be involved are those that normally have a rich deposit of fat, such as over the deltoids and back, on the cheeks, or over the buttocks and calves. When the condition is more widespread the limbs and trunk may become practically immobilised by their rigid subcutaneous casing.

In slight cases the temperature remains normal, but more often it is considerably reduced, and may drop as low as 90° F. Other vital processes also slow down, the respirations may fall to sixteen per minute, the pulse may drop to about sixty per minute, and the output of urine is scanty. Convulsions may occur. There is usually a sharp drop in weight, and pulmonary infections or diarrhœa may occur terminally. There are, however, other instances, usually in slightly older infants, in which sickness and diarrhœa are the primary symptoms, and patchy solidification of the fat appears as a complication. In the localised form recovery is possible if the infant's temperature can be raised and the strength maintained, but the generalised form is almost always fatal.

Studies by Harrison and McNee [1] have shown that the subcutaneous masses are due to deposits of neutral fats in the form of acicular crystals, and that the melting-point of the fat is raised above the normal. The deposits sometimes contain calcium salts, and are then opaque to X-rays, the film showing a speckled appearance of the subcutaneous tissues. Histologically there is an accompanying inflammatory reaction consisting of an invasion of small round cells and a few giant cells. It is uncertain whether the condition is due to a primary error of fat metabolism or to toxæmia damaging the fat cells.

Treatment. There is no local treatment. Care must be taken to keep the body temperature up to normal, stimulant treatment by mustard baths may be required, and every effort should be made to secure breast-feeding.

Scleroderma

This is a rare condition characterised by fibrosis and shrinking of the skin, so that it becomes taut and cannot be creased nor picked up from the underlying tissues. It may occur as isolated patches (morphœa), which resemble pieces of wash-leather let into the skin, but in children it is usually seen in its diffuse form.

In the diffuse form the limbs are chiefly affected, and symmetrically, but the trunk and face may also be involved. At first the skin is slightly swollen, and the onset is often accompanied by feverish attacks, pains in the limbs, and a variety of erythematous rashes. Later the swelling subsides and the skin gradually hardens and contracts so as to restrict very considerably the movements of the joints, the subcutaneous fat disappears, and eventually the affected parts appear emaciated. The skin over the bony prominences becomes stretched, thin, and shiny, and is likely to break down into chronic ulcers, and the fingers become stiff and claw-like (sclerodactyly). Telangiectatic rashes are often present, particularly on the hands, over the cheeks, and round the eyes. The hair becomes thin and may be entirely shed, and extensive erosions of the teeth may occur. Gangrene of the extremities has also been recorded.

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[1] Harrison and McNee, *Arch. Dis. Child.*, 1926, **1**, 63.

The pathological changes consist of fibrosis and atrophy of the skin and subcutaneous tissues. The nutrient vessels of the skin are at first surrounded by a cellular exudation, and later undergo obliterative arteritis. The cause is unknown.

The course is a chronic one over several years. Death may result from exhaustion or intercurrent respiratory infections; in other cases some degree of recovery may come about even after years.

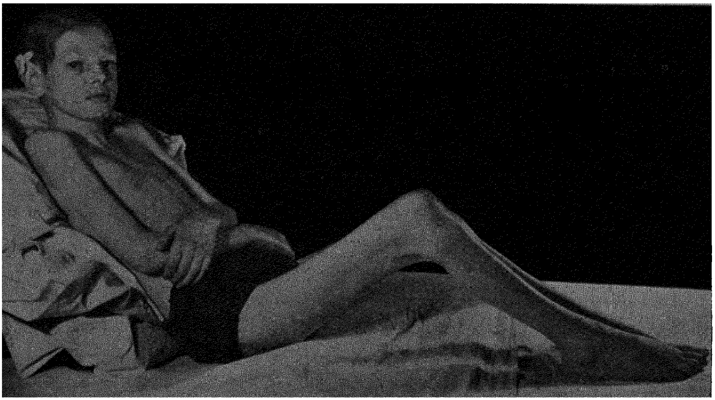


FIG. 126. Dermatomyositis. Boy aged twelve years. The arms and legs show considerable wasting, the knees could only be straightened to the degree shown, and scars of chronic ulcers were present over the patellæ and acromion processes.

Treatment. There is no specific treatment. Septic foci must be sought for and eradicated. Thyroid gland has been recommended, and in the early stages salicylate of soda may be of some value. General measures should include fresh air, a nourishing diet, cod-liver oil and malt, and tonics. Massage and physical therapy may be tried in the chronic stage, and careful splinting may be needed to prevent deformities. There is usually great difficulty in getting the chronic ulcers over bony points to heal; local exposures to ultra-violet light have seemed to help, and dressings of gauze soaked in horse serum have succeeded when the usual antiseptic dressings have failed.

Associated Conditions. Scleroderma may occur alone or may be associated with similar changes in the other supporting tissues of the body.

Muscles. The muscles may be affected in the same way as the skin, becoming small, hardened, and fibrous, to such an extent as to immobilise the joints. If the muscles are affected alone while the skin escapes the name *Myositis Fibrosa* is applied, while if skin and muscle are simultaneously affected the condition is spoken of as *Dermatomyositis.*

Bones. In the chronic stage a considerable loss of calcium occurs from the bones, leading to severe osteoporosis. This may be due to direct interference with the nutrition of the bone, but mere loss of use of the limbs is also a likely factor.

Joints. Fibrosis of skin and muscles may occur together with the changes of rheumatoid arthritis. A boy under the writer's observation began his troubles with an illness indistinguishable from rheumatoid arthritis. Within a year this was complicated by dermatomyositis, and later by calcinosis.

Calcinosis. This name is applied to the formation in the subcutaneous tissues of deposits of calcium salts, chiefly carbonates and phosphates. There may be several subcutaneous plaques, varying in size from a pea to a walnut. They are most likely to appear on the fingers or in the neighbourhood of joints, but may occur in any part of the skin. They form slowly, and may increase until the overlying skin reddens and finally breaks down, allowing the discharge of a milky fluid rich in calcium, or after remaining unchanged for months they may slowly resolve. Rarely a generalised form of subcutaneous calcinosis occurs. The deposits can be seen on an X-ray film.

Pediculosis Capitis

This is a common parasitic infection in children, particularly among those of the poorer classes, and is indicated by the presence of the eggs, or " nits," in the hair. These are small hard white specks which resemble pieces of scurf, but can be distinguished because scurf is easily removed while nits are firmly stuck on to the hair by a chitinous tube, and so cannot be shaken off although they can be made to slide along the hair. The lice themselves are only seen on a close inspection of a heavily infected head. The infection makes the child scratch, and this may lead to a secondary impetiginous infection, the scalp becoming covered with yellow crusts, and the local lymphatic glands becoming enlarged.

Treatment. When there is impetigo the hair must be cut short and the treatment of impetigo adopted. In mild cases the nits may be removed by a thorough washing with soap and water followed by repeated combings with a special nit comb, but if the head is more heavily infected the hair should be soaked with oil of sassafras and the head be wrapped up in a towel overnight. This should be followed by a soap and water wash, and then a thorough combing. The whole process may need to be repeated two or three times before the infection is abolished.

Scabies

This is a common and contagious skin infection caused by the *Acarus scabiei.* Several children in a family are likely to be infected. The acarus burrows into the superficial layers of the skin, the burrows appearing as fine lines about ⅛ inch in length and usually dark with dirt. At one end of the burrow is a minute vesicle containing the acarus, and in order to confirm the diagnosis the burrow may be opened up and the acarus removed on the end of a needle. Under the low power of the microscope the acarus looks like a miniature crab with a fat body and eight short legs. The burrows should be looked for between the fingers and toes, on the front of the wrists, and along the sides of the hands and feet. The skin of the trunk and limbs is often infected as well, but the characteristic burrows are less easily seen there because of the scratch marks and superimposed septic infection. The face is not as a rule involved. Itching is intense, especially at night when the child is warm in bed, and the trunk and limbs may be heavily scored with scratches.

Diagnosis. It is only by bearing this disease in mind in any child who suffers from an itching skin that cases will neither be overlooked nor mistaken for eczema or lichen urticatus. Similar symptoms in other members of the family must always arouse suspicion. It must also be remembered that a septic dermatitis may be masking an underlying infection with scabies. The distribution between the digits and on the wrists is distinctive, for impetigo, eczema, and lichen urticatus do not usually occur in these situations, but the final diagnosis rests on recovering an acarus from a burrow.

Treatment. The two most reliable preparations are sulphur and benzyl benzoate. The sulphur treatment consists of giving a warm bath followed by the inunction of equal parts of sulphur ointment and vaseline; this is repeated for three consecutive nights. The same night-clothes and bedding should be used throughout the treatment so that they may become thoroughly saturated with the ointment, but they should be disinfected after the third night. The sulphur may set up a mild dermatitis, in which case the skin should be swabbed over with a lotion of 1 drachm of liquor picis carbonis to 1 pint of water for a week or so.

The benzyl benzoate treatment is more quickly carried out, and its efficacy is about equal to that of sulphur. The original application, consisting of benzyl benzoate, soft soap, and industrial spirit, was apt to cause a good deal of pain, and the preparation now in use at Great Ormond Street consists of benzyl benzoate 30 parts, methyl cellulose 1·25 parts, water to 100 parts. This makes a non-greasy lotion. The directions for its use are : Allow the child to soak in a hot bath for ten minutes, then gently scrub with soap and water, paying particular attention to the groins, axillæ, wrists, and between the fingers and toes. While still wet apply the lotion with a soft brush to the whole child except for the face and head, rubbing until a lather is obtained. As soon as the lotion has dried, give a second application. Then dry the child and put on the same clothes as were worn before the treatment. Twenty-four hours later, bath the child and put on clean clothes.

Other treatments that have been recommended consist of painting all over with 1 per cent. rotenone following a hot bath ; and painting thrice daily with a solution of 4 oz. of derris dusting powder, 18 drachms of soap flakes, and 1 gallon of cold water.

Whichever treatment is employed, it is essential that the bedding and clothes be sterilised when the treatment is finished. It is equally important that the other members of the household be examined, and those found to be infected be submitted to treatment.

Ringworm (Tinea)

Ringworm in children occurs in two forms—ringworm of the body (*Tinea corporis* or *circinata*) and ringworm of the scalp (*Tinea capitis*). Both forms are due to infection of the hairs and hair-follicles by parasitic fungi.

Tinea Corporis. The fungus may be conveyed from one child to another, but is usually taken from a cat or dog. The condition appears as circular red scaling patches or rings of variable size, either single or multiple, and situated on the face, body, or limbs. The fungus can be seen under the microscope if a scale is removed from the patch and allowed to soak for half an hour in liquor potassæ before examination.

Ringworm of the body clears up in a few days if the patches are painted with tincture of iodine or inuncted with an ointment of salicylic acid gr. 10., benzoic acid gr. 10., coconut oil m. 30., vaseline ℥ss.

Tinea Capitis. The fungus spreads from child to child by contact, or through the medium of brushes and combs, towels, etc. The lesion begins as a single patch, more or less circular, in which the skin appears scaly and the hair seems at first sight denuded. Closer examination shows the stumps of many hairs broken off short or bent over at an angle. The stumps are brittle, and easily break off if an attempt is made to pull them out, but gentle traction will release a stump, and microscopical examination of it when mounted in liquor potassæ will show the fungus. Unless prompt treatment is given fresh patches soon appear, and eventually most of the scalp may be affected.

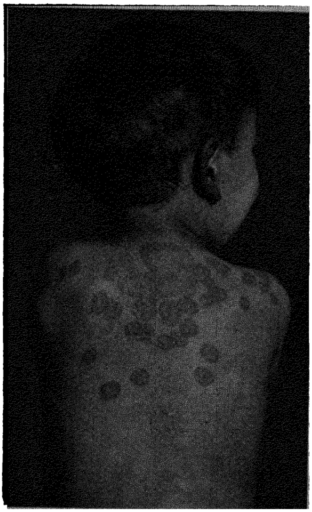


FIG. 127. Tinea corporis. Boy aged three years.

Ringworm of the scalp is more resistant to treatment than ringworm of the body because of the depth of the fungus in the hair follicles. To effect a cure it is necessary to cause shedding of the hair. The older methods of doing this by causing a severe local inflammation with irritant ointments have been superseded by X-ray treatment or the administration orally of thallium acetate. Both methods require caution, and treatment is best left to those with special experience.

With X-ray treatment a single patch is exposed to the rays, or if the whole scalp is involved it is mapped out into five areas, each receiving an equal exposure. Three weeks later the hair falls, and the disease is then cured. New hair begins to appear after another three weeks. The risk of this method lies in producing permanent baldness, although it is not likely to happen in the hands of experts.

The internal administration of thallium acetate is followed by shedding of the hair in one to two weeks. The risk in this case is not of permanent baldness, but unless the dose is accurately measured severe toxic symptoms may be produced, and fatalities have been recorded. One dose of 8 *mgm. per kgm. of body weight* is given. The healthy hair falls first, and then attention must be given each day to the removal of infected hairs by forceps or adhesive strapping.

When suppuration has already occurred before treatment has been given, X-rays and thallium acetate should not be used, but the scalp should be bathed twice a day with hot water, and a 3 per cent. benzoic and salicylic acid ointment be applied.

Ringworm of the nails is rare in children. The nails appear roughened, and scrapings show the fungus. Treatment consists of removing the nail, followed by the application of salicylic and benzoic acid ointment.

Alopecia Areata

This is not uncommon in children, and is characterised by the rapid development of one or more bald patches on the scalp, the skin becoming pale and shiny, There may be a few stumps of broken hairs, which are slender at their junction with the skin, giving them the appearance of exclamation marks.

The cause is unknown. The condition must be distinguished from ringworm, in which the bald patches have a scaly sur-

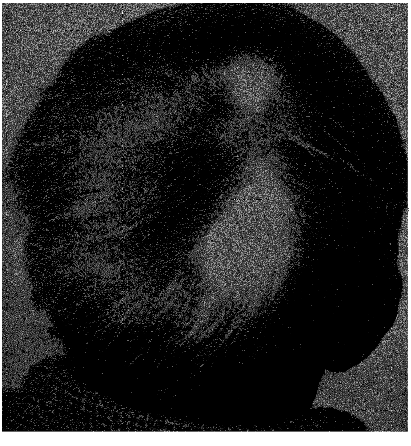


FIG. 128. Alopecia areata. Boy aged seven years.

face, the hair stumps are bent and fragile, and microscopical examination shows the infecting fungus.

Alopecia areata is likely to persist for months or years before new hair grows, and relapses are also common. Treatment consists of giving tonics by mouth, and applying stimulating lotions locally, such as perchloride of mercury ½ per cent. in spirit, or cantharadin in the following prescription :—

Vinegar of cantharadin dr. 3, glycerin dr. 3, phenol gr. 20, almond oil oz. 1½, oil of Rosemary m. 15, water to 6 ozs.

Nævi

Nævi, or hæmangiomata, are for the most part congenital. They fall into four main types :—

(1) *Port-wine Mark.* These may occur on any part of the body, but are most common on the face and neck. The patches are flat, purplish in colour, and may cover extensive areas, sometimes following a segmental distribution. As to treatment, they are best left alone.

(2) *Strawberry Mark.* These vary in size, but seldom cover an area larger than a shilling. They are raised, bright red in colour, and may overlie a deep cavernous angioma. Their treatment depends on their size and position. Small ones may be frozen for about fifteen seconds with a stick of carbon dioxide snow, the application extending just beyond the edge of the nævus. The treatment may require to be repeated three or four times at intervals of a few weeks. Treatment by electrolysis or puncture with the galvano-cautery is also efficacious. Radium may be used for the larger nævi, the best results being obtained if other methods have not been previously tried.

(3) *Cavernous Nœvi.* These are situated in the subcutaneous tissue, and appear as soft raised tumours with a pale purplish tinge. They are best dealt with by surgical excision, although when they occur on a part where a scar would be unsightly, radium may be used instead.

(4) *Spider Nœvi.* These arise after birth, and consist of a bright red vascular twig in the centre surrounded by a network of dilated capillaries. They may be treated by freezing with CO_2 snow, electrolysis or galvano-cautery.

Chilblains (Erythema Pernio)

Chilblains are a frequent source of trouble to children, appearing as red and intensely itching swellings on the toes, heels, fingers or ears. They occur during the cold damp months, and are at their worst when the child warms his cold extremities in front of a fire. In severe cases the surface of the chilblain may ulcerate.

Treatment. Children subject to chilblains should be protected from the cold and damp by wearing thick loosely knitted woollen stockings and gloves. Daily exercise to promote a good circulation, such as skipping and swinging the arms, should be encouraged. Locally a stimulating application such as liniment of camphor should be rubbed in, and as a preventive measure the extremities may be rubbed with camphorated oil every morning and evening. If ulceration occurs a dressing of boracic ointment should be applied. Internal remedies are of doubtful value. Calcium has been much vaunted, and may be given in the form of 10 gr. of calcium lactate in a simple mixture three times a day. The results of calcium therapy are, however, not impressive.

Warts

These consist of small epithelial excrescences, and are mildly infectious. Two varieties are met with—the common wart and the plane wart, both due to a virus infection.

The *common wart* may be either single or multiple, and has a surface which is ragged, often lobulated, and usually darkened by dirt. They occur most frequently about the fingers or on the backs of the hands, and, even if left untreated, may disappear spontaneously after months or years. Children are very apt to pick at them, causing a troublesome oozing of blood.

Plane warts consist of circular flat-topped pale growths not much larger than a pin's head, and are usually very numerous. They occur chiefly on the hands, forearms, and face.

Treatment. Large solitary warts may be treated by the direct application of a drop of fuming nitric acid off the end of a match-stick, the surrounding skin being carefully protected by a coating of vaseline. Multiple warts may be dealt with by freezing with a stick of CO_2 snow, or by a short exposure to X-rays, or by the application of tri-chlor-acetic acid. This is a crystalline substance; one drop of water should be added to some of the crystals to dissolve them, and the fluid applied in the same way as fuming nitric acid.

CHAPTER XXV

CONGENITAL SYPHILIS

THE vast majority of children suffering from syphilis are infected before birth, and it is quite exceptional for the condition to be acquired during childhood. This may, however, come about if an infant is suckled by a wet nurse who has syphilis, or perhaps through the medium of infected utensils used for feeding or toilet purposes. The sequence of events is then the same as in adults—after about a month or six weeks a primary sore appears at the site of infection, with firm enlargement of the regional lymph glands, followed in two or three months by a secondary stage in which a variety of symptoms, such as skin rashes, ulceration and condylomata around the mouth and the anus, faucial and laryngeal catarrh, and a general enlargement of lymph glands, may appear. The tertiary stage follows after a lapse of a few years, and is characterised by a diffuse fibrosis of organs or by the formation of gummata.

The fœtus may be infected by either parent, but as a rule the infection takes place from the mother *viâ* the placenta. It is, however, possible for the father directly to infect the embryo if the seminal fluid contains spirochætes. In either case the mother will be infected by the time her child is born, and in practice it is found that the Wassermann reaction of the woman who bears a syphilitic child is almost invariably positive, even though she herself may not show any clinical evidence of syphilis. That she is already infected is also indicated by Colles' Law, which states that the mother of a syphilitic infant may breast-feed her child without risk of infecting herself.

Because the great majority of syphilitic infants acquire their infection during intra-uterine life, a primary stage is not apparent. If the infection begins shortly after conception the fœtus will almost certainly die *in utero* and be cast forth as a miscarriage or still-birth. If infection is delayed, the infant may be born prematurely, and at birth may show obvious signs of secondary syphilis, in which case death is likely after a few days or weeks. It more commonly happens, however, that the child is born alive and apparently healthy, and manifestations of the disease are delayed for a month or so. The symptoms that then arise seem to be a mixture of secondary and tertiary lesions, illustrated on the one hand by skin rashes and condylomata, and on the other hand by inflammation of bone and pericellular cirrhosis of the liver. The clinical demarcation of the secondary and tertiary stages is much less clearly defined in congenital syphilis than in the acquired form of the disease.

The question has often been raised whether syphilis can be transmitted through three generations. There is considerable difficulty in proving this, because although a syphilitic baby may be born to a parent who also shows obvious stigmata of congenital syphilis, it is, of course, possible that he or she may have acquired a fresh infection in adult life or that the other parent may have done so. In spite of these difficulties it seems probable that in a small number of cases syphilis does pass on to the third generation, and recently Nabarro [1] has recorded 17 families illustrating third-generation syphilis.

Symptoms in Infancy. When signs of syphilis are present at birth the infant is usually undersized and puny, with a wrinkled skin and wizened face. Nutrition seems to fail right from birth, and after a month or so the baby appears miserably thin and wasted. On the other hand, if the symptoms are delayed for a few weeks growth may proceed satisfactorily enough, and the infant may be well nourished when the first manifestations appear.

Syphilis in infancy may produce a multitude of symptoms, many of which may be present at the same time, or they may occur singly. One of the most frequent is snuffles, due to rhinitis. The nose may become so blocked from congestion and discharge as to interfere seriously with breathing—and therefore with feeding and nutrition, because the infant continually has to turn from the nipple in order to breathe through the mouth. Snuffles may be present at birth or may appear after a few weeks, and the accompanying necrosis of the nasal cartilages and bones accounts for the depressed bridge of the nose which so frequently characterises older syphilitic children. Ulceration of the larynx may also occur, giving a hoarse character to the infant's cry.

Skin rashes are frequent, but vary considerably in appearance. As a rule the basis of the rash is a blotchy erythema, usually dull red in colour, and eventually the superficial layers of the skin flake off in small scales. Although almost the whole skin surface may be involved in this way, the rash and subsequent flaking are particularly common over the buttocks and on the soles of the feet and palms of the hands, where the skin may come to present a highly glazed appearance. There is often difficulty

[1] *Brit. Jour. Ven. Dis.*, **1933, 9,** 1.

in distinguishing the rash on the buttocks from a simple napkin rash, but while the latter tends to affect particularly the prominent parts of the skin which come into firm contact with the napkins, the syphilitic rash is often more pronounced in the creases of the skin and close up to the edge of the anus. Linear cracks or ulcers are likely to appear, radiating from the anus like the spokes of a wheel, and there may also be raised greyish masses of epithelium, or condylomata, close to the anal margin. Similar patches and linear ulcerations may occur round the corners of the mouth, and later give rise to radiating scars called "rhagades." Sometimes the skin may be raised into blisters filled with thin sero-purulent fluid—syphilitic pemphigus—and the outlook is then always grave. Another occasional manifestation is the formation of large hard boils, which, if they discharge at all, discharge only a thin fluid. Inflammation of the nail beds may also occur—syphilitic onychia—often with loss of the nail. Anæmia is a common feature, and accounts in part for the *café-au-lait* complexion which many of these infants show. The blood count shows a simple secondary anæmia, in which both the number of red cells and the hæmoglobin are reduced.

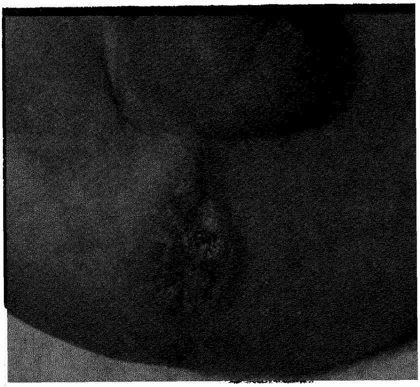


FIG. 129. Syphilitic condylomata around the anus. Infant aged two months.

Visceral disturbances vary a great deal. The liver is usually enlarged, extending perhaps almost to the umbilicus. It is firm, with a smooth surface, and not tender. As a rule jaundice is not present, but when it occurs it indicates an unusually severe infection, and carries a bad prognosis; it is most likely to be seen in those infants who show manifestations of the disease right from birth.

At post-mortem examination the liver is reddish-purple in colour, or may be slightly jaundiced, and histologically a fine network of fibrils of young fibrous tissue can be made out between the hepatic cells—pericellular cirrhosis. There may be also numerous small foci of necrosis with collections of lymphocytes, representing miliary gummata. Special staining methods show an abundance of spirochætes.

The spleen is also usually enlarged, and may extend about two fingersbreadth below the costal margin. It feels smooth, but is noticeably firm. That the kidneys may also be involved is indicated by the occasional presence of albumin, casts, and blood cells in the urine, and sometimes a generalised œdema.

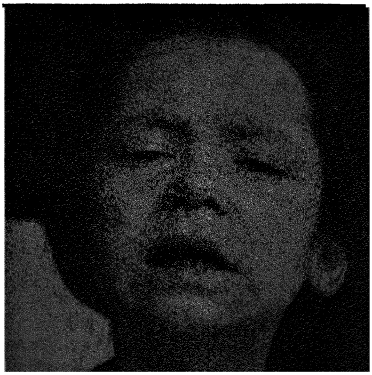


FIG. 130. Syphilitic scars round the mouth—" rhagades."

Infection of the lungs—pneumonia alba—has been described in young infants, but is very rare. The lungs are heavy, and show areas of pale tough consolidation which histologically consist of a great increase of fibrous tissue. Orchitis is another manifestation which may appear shortly after birth, and gives rise to a hard but painless swelling of the testicle. This is of clinical value because it is easily recognised, and may suggest the correct diagnosis. Ocular manifestations at this age include iritis and choroiditis.

Involvement of the nervous system may be indicated in various ways. There may be frank signs of meningitis, with stiffness of the neck, Kernig's sign, and perhaps convulsions. The meninges at the base of the skull may be particularly affected, which accounts for a varying degree of hydrocephalus, as shown by the gradual increase in the size of the head and by the distension of the scalp veins. The cerebro-spinal fluid is under increased pressure, and may contain an excess of lymphocytes up to about 100 per c.mm., the globulin is increased, and the Wassermann reaction is positive. With energetic and persistent treatment the nervous manifestations of infancy may sometimes resolve completely, but spastic paralysis, mental defect, fits, and evidence of pituitary disorder may appear as sequelæ.

Involvement of the long bones is, in infancy, one of the most common effects of syphilis, although clinical evidence of it is usually delayed until the second or third month after birth. The upper end of the tibia is most frequently affected, but any of the long bones may be involved, and the condition is so painful that the infant may refuse to move the limb—hence the name syphilitic pseudo-paralysis. Examination may show some slight swelling, with considerable tenderness, towards the ends of the long bones, and in severe cases there may be actual separation of the epiphysis with deformity of the joint outline. X-ray examination shows a typical picture, in fact characteristic radiological changes may appear very shortly after birth, several weeks before there is clinical evidence that the bones are involved. The film shows an irregular epiphysial line, with excavations running back towards the shaft of the bone as though pieces of the young bone had been nibbled away. In addition there is usually evidence of periostitis along the shafts of the bones (see Plates XIII. and XIV.).

Syphilitic pseudo-paralysis may be the first and only sign of congenital syphilis, or it may be added to a history which already includes snuffles and perhaps rashes on the buttocks and hands and feet. The diagnosis of syphilitic epiphysitis is seldom difficult, for although pain and loss of movement are also features of infantile scurvy, the age incidence is different, syphilis appearing within the first six months of life while scurvy occurs in the second half of the first year ; nor should confusion arise between syphilis and the multiple acute suppurative arthritis of infancy, for in the latter there is high fever and grave constitutional disturbance, together with swelling and tenderness confined to the joints, and without corroborative evidence of syphilitic infection.

Of other skeletal lesions, dactylitis occasionally occurs. Generally the proximal phalanx of one or more digits is affected, causing a fusiform swelling. An X-ray shows the presence of osteoperiostitis. Syphilis may also affect the skull bones, sometimes causing areas of rarefaction by the formation of multiple gummata, but more often giving rise to a gradual condensation of the bone, so that the skull eventually becomes much thickened.

Late Congenital Syphilis. After the rush of symptoms that characterises syphilis in infancy the disease enters upon a quiet phase which continues up to about the sixth year, when manifestations of late congenital syphilis begin to appear. During the quiet phase, however, such symptoms as hæmaturia, dactylitis, or convulsions may arise from time to time to indicate that the infection is slowly progressing.

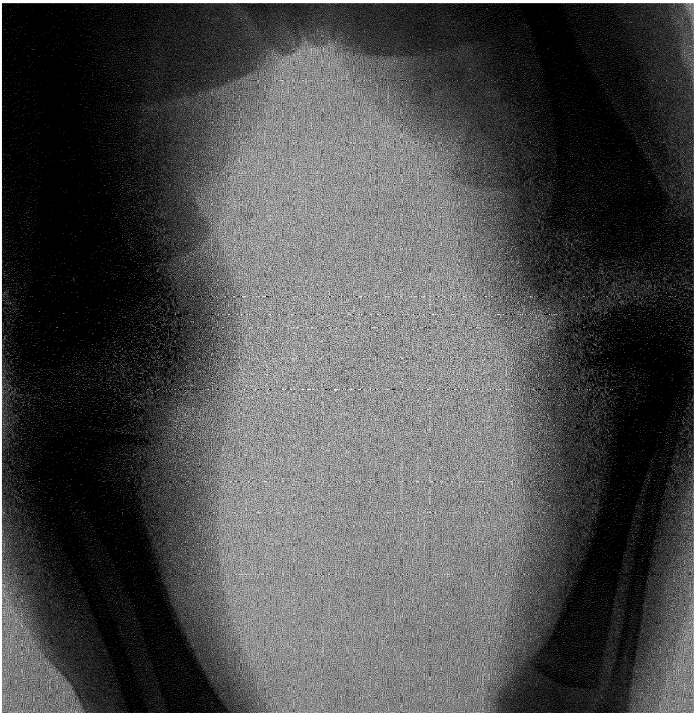
The symptoms of late congenital syphilis include interstitial keratitis, changes in the teeth, deafness, chronic periostitis, and more rarely cirrhosis of the liver, juvenile general paralysis, and juvenile tabes. Unlike the later lesions of acquired syphilis, disease of the heart and large vessels is very rare. The general effect is to interfere with the nutrition of the child, leading to stunting of height and a low weight. Anæmia is usually present, and gives a sallow complexion. A general enlargement of the superficial lymph glands has sometimes been attributed to late syphilis.

Interstitial Keratitis. This begins as a small pinkish-grey patch near the margin of the cornea—the so-called " salmon patch " —which gradually spreads until the whole cornea is involved. When this stage is reached the cornea appears steamy and opaque, and there is considerable congestion of the conjunctiva. This state of affairs lasts for a few weeks and then the cornea slowly begins to clear, and after about three months the condition has usually resolved completely, but small opacities may remain permanently. Almost always both eyes are involved, one beginning a few weeks later than the other. Interstitial keratitis is a peculiar manifestation in that it may appear even while the child is undergoing intensive treatment, and the course of the keratitis is not appreciably influenced by treatment. In severe cases there may also be iritis, which may be followed by distortion of the pupil owing to the formation of adhesions.

The importance of examining the eye for choroido-retinitis in any child suspected of syphilis must be emphasised, for this may be the only confirmatory evidence of a suspected syphilitic infection. The choroiditis is a disseminated lesion in which numerous white and black patches are dotted about the fundus, The larger patches are found towards the region of the macula. while at the periphery they appear only as a fine dusting of black pigment.

The Teeth. The first dentition shows no alterations characteris-

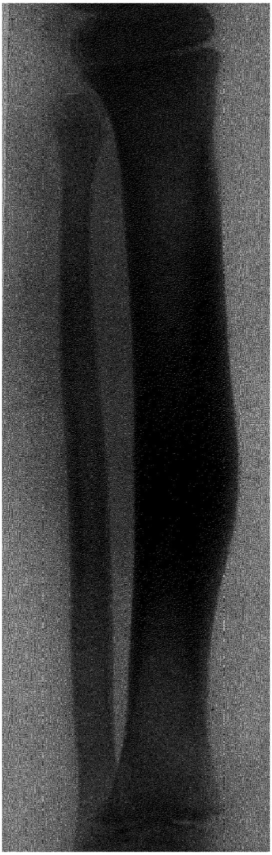
PLATE XIII.



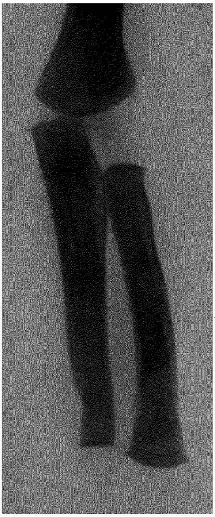
Syphilitic osteo-periostitis in an infant aged three months. Note the excavations at the upper ends of the tibiæ, and the slight periostitis along the shafts of the tibiæ and femora. (By courtesy of Dr. Shires.)

[*To face p.* 654.

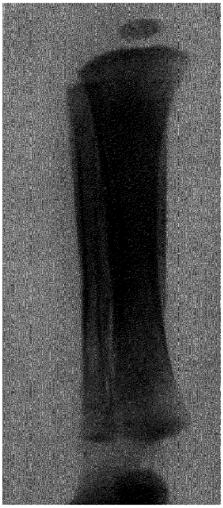
PLATE XIV.



(A) Syphilitic osteo-periostitis of the tibia (*sabre tibia*) in a boy aged seven years.



(B)



(C)

(B and C) Syphilitic osteo-periostitis of the radius and ulna (B), and tibia and fibula (C), in an infant aged five months.

[*To face p.* 655.

tic of syphilis. Hutchinson's teeth affect the incisors of the second dentition ; they are broader at the base than at the cutting edge, and in the centre of the cutting edge there is an unmistakable notch. Although Hutchinson's teeth are of great diagnostic value, they only occur in about one-quarter of the cases. The six-year-old molars may also appear dome-shaped (Moon's molars).

The Bones. The effect on the bones is to produce a sclerosing type of osteitis and periostitis, so that the bones become much heavier than normal. This change chiefly affects the skull and the long bones, and is often best illustrated in the tibia, where the middle of the shaft is particularly thickened and the bone tapers towards the lower end like a sabre (sabre tibia : see Plate XIII.). There may be a good deal of aching in the affected bones, especially at night when the child is in bed. In untreated cases gummata may form in the bones and later break down to leave a chronic discharging sinus, and similar changes may occur in the skull. Necrosis of the nasal bones and of the hard palate is not uncommon, the former leading to flattening and depression of the nasal bridge and the latter to perforation into the nose. There may be a highly offensive nasal discharge with the formation of big crusts in the nose.

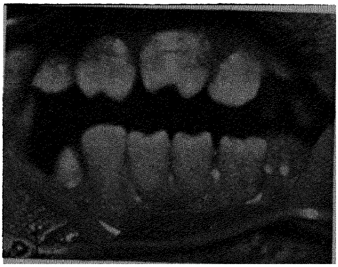


FIG. 131. Hutchinsonian teeth in congenital syphilis. From a girl aged ten years. (By courtesy of Dr. Nabarro.)

Joints. Painless effusion into the synovial cavities of the joints may occur, and is most likely to affect both knees (Clutton's joints). The effusion rapidly absorbs under treatment. A multiple affection of the small joints resembling rheumatoid arthritis has also been recorded.

Visceral Changes. The liver may gradually become much enlarged, extending almost to the umbilicus. It is firm on palpation, the edge is rounded off, and the outline may be notched and irregular owing to the formation of gummata and extensive scars. Neither ascites nor jaundice are usual. Under treatment the liver sometimes becomes gradually smaller, although it is difficult to decide whether this really marks an improvement or whether it is due to an increased contraction from fibrosis. The spleen is also enlarged in company with the liver, and may reach to the umbilicus ; it is firm on palpation and not tender. The enlargement is associated with considerable fibrosis, but the formation of gummata in the spleen rarely occurs. There is sometimes evidence that the kidneys are involved, as is shown by persistent albuminuria and the passage of casts with occasional attacks of hæmaturia. While in infancy the effect on the kidney is to give rise to symptoms of acute nephritis, in later childhood the symptoms are caused by progressive renal fibrosis. Recurrent attacks of paroxysmal hæmoglobinuria may occur, and in childhood are almost always associated with a positive Wassermann reaction. The urine is a bright red colour and gives positive chemical tests for blood, but microscopical examination fails to show any red cells. As in adults, these attacks may be provoked by exposure to cold. They are quickly abolished under treatment.

In the later years of childhood progressive deafness may slowly develop, owing to disease of the auditory nerve. Fortunately it is not a common symptom, as it is uninfluenced by treatment.

Neurosyphilis. The late nervous manifestations of congenital syphilis include juvenile general paralysis, juvenile tabes, and a considerable variety of symptoms produced by vascular disturbances, mostly thrombotic. Thus a single cranial nerve may be picked out, or there may be a more extensive paralysis or repeated convulsive attacks. Under treatment,the effects caused by vascular syphilis may improve to a great extent, but there is usually some residual paralysis. Occasionally there is nothing in the previous history to suggest syphilis—this was so in a child of eleven years who had always seemed in perfect health until one day at a cinema she complained of giddiness. An hour later she became unconscious, and on regaining her senses some nine hours later was found to have a right-sided hemiplegia and hemianæsthesia. The Wassermann reaction was strongly positive. Ultimately she was left with a slight spasticity of the right arm and leg.

While in adults tabes dorsalis is more common than general paralysis, the reverse is the case in congenital syphilis. Neither form of juvenile neurosyphilis is common before the beginning of the second decade, although one child with general paralysis under the author's care was only four years old. The clinical appearance and cerebro-spinal fluid changes are identical with those seen in adults except that, as one would expect, the mental perversion in juvenile general paralysis takes on a childish form ; the young patient loses all trace of shyness, and becomes loquacious, mischievous, dirty, or morally delinquent. The course of the disease is slowly downhill.

Diagnosis. A Wassermann reaction should be carried out in all cases where there is ground for suspecting syphilis, and indeed it should also be performed even when the clinical manifestations leave little room for doubt, for the test will then give some guide as to the effect of treatment. During the first few weeks of life the Wassermann reaction is less reliable than later on, and therefore it is advisable to test the mother's reaction as well as that of her infant, at any rate until the baby is three months old, because the Wassermann of the mother of a syphilitic child is almost always positive. In older infants the reliability of the Wassermann is very considerable—it is positive in about 96 per cent. of children who at the time of the test show clinical evidence of the disease, while when clinical signs are dormant the test is positive in about 80 per cent. When the test is being carried out on account of symptoms derived from the nervous system, it should be made on the cerebro-spinal fluid as well as the blood.

In recent years a flocculation test for syphilis—the Kahn test—has been employed. Although it is simpler to carry out than the Wassermann reaction, it does not give a positive result in quite such a high percentage of cases, and therefore the Wassermann is the test to be preferred.

Of the early symptoms, snuffles and rashes are the most common, but snuffles by itself is not always reliable evidence of syphilis. It is, for instance, very common in mongol babies, and occurs when there is a congenitally large pad of adenoids, or may be caused by a simple cold. The rashes take such a diversity of form that it is often difficult to be sure of their significance without the aid of a Wassermann test. Confusion is particularly likely between a syphilitic rash and a simple napkin rash, although attention has already been drawn to the slight differences between them. In young infants X-ray examination of the long bones is of great diagnostic value, for changes may appear in the film very soon after birth, and certainly some weeks before there is clinical evidence of osteo-periostitis.

Prognosis. When signs of active syphilis are present in the newborn child, the outlook is always grave, and in spite of treatment many of these infants die within a few weeks ; on the other hand, when the signs are delayed for two or three months the prognosis under treatment is good as regards life, and many of these children go on to ultimate cure. In others, although life continues, lesions such as depression of the nasal bridge, deafness, corneal opacities, or choroiditis may be permanent witness of the syphilitic taint. The treatment of late neurosyphilis—general paralysis and tabes—is as unsatisfactory as it is in adults. Energetic treatment may very occasionally lead to a cessation of the infection, but the damage already done to the nervous system remains.

Treatment

The employment of spirochæticidal drugs is the essence of treatment, and the three drugs generally used are arsenic, mercury and bismuth. As a rule a course of arsenical injections is given in combination with, and followed by, a long course of treatment with mercury. Bismuth is not so strongly spirochæticidal as arsenic, but is more potent than mercury. Bismuth and mercury should not, however, be given together, as their toxic symptoms are the same and therefore there is a risk of a cumulative effect.

Treatment of Infants. *Arsenic.* Arsenic may be given intravenously, intramuscularly, or by mouth, different preparations being used according to the route selected. Although intravenous administration enables the most powerful spirochæticides to be given, this method has obvious technical difficulties in infancy, as should it happen that any of the drug be inadvertently put into the subcutaneous tissues outside the vein, a severe local reaction is likely to occur, and may lead to sloughing. For intramuscular injection sulph-arseno-benzene (sold under such names as Sulpharsenol, Kharsulphan, Sulphostab, Sulpharsphenamin) should be used. It is readily soluble in sterile water. A course of eight weekly injections should be given, the first dose being not more then 0·02 gm., and this may be gradually increased to a maximum of 0·12 gm. The dose must, however, be graduated according to the condition of the baby, and if the infant is very puny and wasted, as weak a dose as 0·001 gm. may be given initially. The injection is made deeply into the gluteal muscle at a point half-way between the anterior superior iliac spine and the lower end of the sacrum. The course should be repeated in three months' time, beginning with a dose of 0·06 gm. and working up to 0·2 gm.

Arsenic by mouth, in the form of Acetarsone (Stovarsol)[1] gives satisfactory results in infancy, although for older children the stronger preparations for intravenous use should be employed. A course consists of seven periods each of ten days, with a four-day interval between each period. For feeble infants, and those whose weight is below 10 lbs., the number of tablets per day in successive periods should be ½, 1, 1½, 2, 3 ,4, 4, while for infants over 10 lbs. the dose should be 1, 1½, 2, 3, 4, 4, 4.[2] As far as is practicable, the daily amount should be divided into three or four doses. A second course may begin after an interval of three months. Toxic effects, such as dermatitis and diarrhœa, must be watched for, especially in feeble infants, and, should they appear, the drug must be stopped for a few days.

Whether arsenic is given intramuscularly or by mouth, it should be accompanied by the daily inunction of mercury ointment, and a Wassermann reaction should be performed about a month after each course. Even if the first course is followed by a negative reaction, it is generally wise to give a second course.

Mercury. Mercury should be combined with arsenic. Although it is a less effective spirochæticide, there is no doubt that the symptoms of syphilis in infancy can be entirely got rid of with mercury alone, but a permanent cure is much less likely, and fresh manifestations may crop up in later childhood. The drug may be given by mouth or by inunction. The usual preparation by mouth is Hydrargyrum c̄ Creta ½–1 gr. twice a day. It is as well to combine it with 2 gr. of aromatic chalk powder to prevent diarrhœa, or ¼ to ½ gr. of pulv. ipecac. co. may be used instead for this purpose. If diarrhœa is persistent, the dose of mercury must be omitted for a few days. At the same time, mercury in the form of blue ointment should be rubbed daily into the skin. In order to prevent mercurial dermatitis a different area of skin should be used each day, either the skin of the trunk or the inner aspect of the arms being a convenient site. The ointment should be applied at night-time, and any remains of it wiped away next morning. A watch must be kept for toxic symptoms from the drug, such as sialorrhœa or gingivitis. In the absence of these the administration of mercury should continue for at least a year.

The proper care of syphilitic infants implies a careful follow-up of each case. After the second course of arsenical injections is completed, and while mercury is still being given, the child should be seen at three-monthly intervals for the next two years, and at six-monthly intervals for a further two years, the Wassermann reaction being tested twice a year. A recurrence of symptoms or a reversion to positive in the Wassermann reaction would be an indication for further treatment.

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[1] Stovarsol is prepared by May and Baker in tablets containing 4 gr. (0.25 gm.).

[2] Findlay, L., *The Practitioner*, 1942, **148,** 287.

The Treatment of Older Children. The treatment of older children follows the same principles as in infancy. After the second or third year arsenic can be given intravenously more easily than in infancy, and for this purpose neosalvarsan, "914" (trade names Novarsenobillon, Neokharsivan, Novarsphenamin) may be used. For a child of from three to six years of age the initial dose should be 0·1 gm., and this should be increased over a course of six injections to 0·2 gm., while for children of ten or twelve years of age the dose may be raised to 0·3 gm. The drug is dissolved in 2 or 3 c.c. of sterile water, and the injection is made into one of the veins at the elbow. The vein can be easily made prominent by an assistant compressing the arm just above the elbow, which is less worrying to the child than a tourniquet, and also enables the arm to be held still. Great care must be taken to see that none of the injection escapes into the surrounding tissue, otherwise severe inflammation will follow ; if the slightest swelling appears during the injection, an attempt should be made to withdraw some of the fluid into the syringe, and then the injection must stop. 3 c.c. of sterile saline should then be injected into the part where the swelling has appeared, and the area should be gently massaged.

Occasionally in older children intravenous injections are technically difficult, and then resort must be made to intramuscular injections. The dose of sulph-arseno-benzene (or its equivalents) at five or six years of age would be 0·06 gm., increasing up to 0·3 gm.

While arsenic is being given, a careful watch must, of course, be kept for toxic symptoms such as albuminuria or jaundice, and, should either of these appear, the injections must at once be discontinued. Mercury should be given by mouth and by inunction concurrently with the arsenic. A preparation of pentavalent arsenic—tryparsamide—has been recommended for neurosyphilis, and may be given either intravenously or intramuscularly. A course of eight injections should begin at a dose of 0·5 gm. and increase to 2 gm. It is not, however, so strong a spirochæticide as the other preparations of arsenic. Treatment with malaria, and repeated protein shock, have also been used in juvenile general paralysis, but with disappointing results.

Bismuth. Bismuth is usually given by intramuscular injection. A convenient preparation is bismostab, which is an insoluble form of bismuth suspended in isotonic glucose solution. A special strength is prepared for children, containing 0·2 gm. of bismuth in 5 c.c. In infancy the dose should be three to four minims given as an intramuscular injection twice a week over a period of six to eight weeks. For the treatment of late congenital syphilis the adult preparation of bismostab should be used. Signs of toxicity from bismuth must, of course, be watched for; they include stomatitis, anæmia, and loss of weight.

In addition to specific drug treatment, some of the symptoms call for special management. The snuffles of infancy may be temporarily relieved by using the following oil: menthol gr. 1, eucalyptus oil m. 2, liquid paraffin to 1 oz. One drop of this oil should be instilled into each nostril a few minutes before a feed is due. Breast milk is of great value for syphilitic infants, in fact the possibility of obtaining breast milk directly influences the prognosis. For the anæmia, iron and ammonium citrate (gr. 3) or reduced iron (gr. 1) should be given three times a day.

Prevention. The prevention of congenital syphilis lies in treating the mother during her pregnancy. This is accomplished by combining injections of arsenicals with mercury by mouth, and provided that treatment begins in the early months of pregnancy the birth of a healthy infant with a negative Wassermann reaction can be confidently expected, and the child is likely to remain free from manifestations in later years. Treatment of the mother should be repeated in later pregnancies, even if her Wassermann is negative, in order to ensure the birth of a healthy child. Of congenital syphilis it may certainly be said that prevention is better than cure, for it is more easily carried out, and is more certain of attaining its object.

CHAPTER XXVI

ACUTE INFECTIOUS FEVERS

Diphtheria

DIPHTHERIA is the result of infection by the bacillus diphtheriæ, first isolated by Klebs and Loeffler. The organism produces a local reaction at the site of its implantation, which is most commonly on the back of the throat. Here it gives rise to the formation of a tough membrane composed of fibrin, leucocytes, epithelial cells, and organisms. At this local lesion the bacilli produce an exotoxin, which, reaching the circulation, may cause toxic effects in other parts of the body, particularly on the heart muscle and in the central nervous system. Much of the danger of diphtheria lies in these toxic effects. The local lesion may of itself prove fatal when situated in the larynx or trachea, the membrane obstructing the airway and causing death by suffocation. Infection of the local lesion with secondary invaders, notably streptococci, may also account for serious complications.

With the discovery of diphtheria antitoxin by Behring and Roux, the mortality of the disease has been very considerably reduced. More recently, wherever the "active" immunisation of the child population has been carried out on a large scale (see later under Prophylaxis), the effect has been to diminish materially the incidence of the disease.

Etiology. Diphtheria is essentially a disease of childhood, reaching its greatest incidence between the second and tenth years. It is very uncommon during the first year because the infant obtains a certain degree of immunity from the mother. The disease is most prevalent in the autumn and winter months.

Diphtheria is most commonly spread from one person to another by droplets of infected sputum, for example while coughing, sneezing, or talking. The person carrying the infection may of course be suffering himself from the disease, or he may be harbouring virulent organisms in his nasal and throat secretions while he himself is immune and therefore free from symptoms; such a person is named a carrier. Outbreaks of the infection have also been traced to milk, infected by a human carrier.

The incubation period is between one and five days.

Symptoms. Faucial diphtheria begins in the same way as does an ordinary sore throat. Older children may complain of soreness and pain on swallowing, but younger children seldom do so, and in them such general symptoms as fever, irritability, refusal of food, or drowsiness may mark the onset. Vomiting is usual, although not quite so constant a feature of the onset as it is in scarlet fever. The throat at first appears reddened and injected, and after a few hours small greyish-white spots of exudate appear on one or both tonsils. After twelve hours or so these have spread and coalesced to form a membrane, which at first covers the tonsil and from there soon spreads over the pillars of the fauces, the soft palate, uvula, and pharynx. The membrane is typically greyish-white in colour and adheres to the tissue beneath so that it cannot easily be rubbed off, and if forcibly picked off leaves behind a raw bleeding area which in a few hours may become coated again with fresh membrane. The amount of inflammatory reaction in the throat varies considerably. There are mild cases in which the general disturbance is but slight, the amount of exudate may be insignificant, and unless a culture is made from a throat-swab the true condition may be easily overlooked. More often the faucial pillars are swollen and œdematous, the cervical glands rapidly become enlarged and tender, and in severe cases there may be so much cellulitis that the child appears to have a thick collar of swollen tissue around the neck, and the face becomes bloated and pale. The breath soon becomes offensive, and there may be a profuse purulent or blood-stained discharge from the nose.

The temperature is raised three or four degrees, but the height of the fever is no indication of the severity of the infection ; indeed in the worst cases the child may be so rapidly prostrated that the temperature is hardly raised at all. The pulse is rapid and soft, and there is usually a thick cloud of albumin in the urine. Very rarely a hæmorrhagic form of diphtheria occurs, characterised by much sanguineous discharge from the nose and throat, profound toxæmia, and purpuric hæmorrhages into the skin. The outlook in this form is very grave, and serum treatment seems to be without effect.

The membrane usually reaches its maximum after four or five days, and by that time, unless treatment has been given, the child appears severely toxic. There is a considerable degree of anæmia, the pulse is rapid and of low pressure, and there may be either coma or a muttering delirium. If recovery takes place the membrane now begins to separate, becoming brown and shrivelled, and finally is cast off in masses of varying size.

Laryngeal Diphtheria

Diphtheritic laryngitis is more common in children than adults, and is usually the result of a spread of infection downwards from the fauces. The membrane may descend still lower and form a cast of the trachea and main bronchi.

Symptoms. There may be a history of a sore throat for a day or two beforehand, but often the first symptoms are those of acute laryngitis. The voice becomes hoarse, indistinct, and is eventually lost, and there may be a good deal of coughing with a barking character. The temperature is high, and the pulse and respiration rates steadily rise. As the amount of membrane increases and the airway becomes narrower, suffocative attacks occur in which the breathing becomes croupy or stridulous, and the child appears cyanosed. These attacks become more and more frequent until finally they are practically continuous, and breathing is then laboured and noisy, the child appears livid, and with each inspiration the episternal notch and the intercostal spaces are indrawn. Unless relief by tracheotomy or intubation is promptly given, death ensues from asphyxia.

Nasal Diphtheria

This also arises as a rule by direct spread from a faucial infection, but occasionally the infection begins in the nose. The symptoms are those of nasal obstruction, together with a discharge which is bloodstained, and the skin below the nostril is frequently excoriated. One or both sides of the nose may be affected. The cervical glands are generally enlarged, but toxic manifestations are less common in nasal than in other forms of diphtheria. On the other hand, nasal infection is very likely to persist, and accounts for a large number of diphtheria carriers.

Diphtheritic infection in other parts of the body is uncommon. The middle ear may be directly infected from the throat and give rise to an infectious discharge, and diphtheria of the conjunctiva and of the vulva and umbilicus is occasionally met with.

Complications. The two most important complications are post-diphtheritic paralysis, which has already been described in the chapter on Nervous Diseases, and cardiac complications.

Diphtheria may affect the heart in one of two ways. The circulating toxin may directly damage the heart muscle, causing fatty degeneration of the muscle fibres, or the nervous control of the heart may be interfered with through the effect of the toxin on the cardiac nerves.

The evidence of cardiac involvement lies in dilatation of the heart and weakening of its action, and signs of these are likely to appear at any time from the end of the first week up to one month. The child becomes increasingly pale and restless, and is usually sick. The pulse rate increases, the apex beat moves outwards and becomes less defined, the first sound at the apex becomes short and sharp, giving a tic-tac quality to the sounds, and the blood pressure falls. The danger of diphtheritic carditis lies in the fact that myocardial failure may come on suddenly and unexpectedly, sometimes causing the sudden death of the child, and it is because of this that diphtheritic patients should be nursed absolutely flat for at least a month, until it is certain that the myocardium has escaped. At other times myocardial failure is less dramatic in its onset, but it may be none the less dangerous, and the various evidences of failure such as breathlessness, increasing size and tenderness of the liver, and œdema must be taken to indicate a progressively downhill course. Disorders of cardiac rhythm may also occur, such as extrasystoles, paroxysmal tachycardia, and partial or complete heart block. Of these, extrasystoles are the most common and may persist for years. Rarely an intra-cardiac thrombus forms, and may give rise to emboli. The rare instances of hemiplegia associated with diphtheria are attributed to cerebral embolism.

Endocarditis as a complication is rare, but every now and again rheumatic heart disease has its origin in an attack of diphtheria. This was so in 4 out of 250 consecutive cases of rheumatic heart disease. It is probable that when rheumatism follows diphtheria it is due to secondary infection in the throat, probably streptococcal, rather than to the diphtheria itself. Bronchopneumonia is fortunately not a common complication. It is most likely to arise in laryngeal cases, particularly if tracheotomy has been necessary. Nephritis is also uncommon, although albuminuria is usual during the acute stage, the amount of albumin varying with the severity of the faucial inflammation. Suppuration of the cervical glands may arise during convalescence.

Diagnosis. An early diagnosis of diphtheria is of the greatest importance because the administration of anti-toxic serum is a life-saving measure, and the benefit of the serum becomes less as each day passes. If there is any reason to suspect that a sore throat is diphtheritic, serum should be given at once without waiting for the report of the throat swab, for to delay treatment may allow the membrane to spread to more dangerous places such as the larynx, and also the chance of preventing toxic complications becomes less.

A proper examination of the throat is essential, and care should be taken that the first view of the throat is complete and sufficient. The child must be facing a good light, and should be restrained from interfering with the examination. The presence of greyish-white membrane on one or both sides of the throat, with fœtor of the breath, nasal discharge, and swollen cervical glands, would leave no doubt of the diagnosis, but in an earlier case the membrane may be slight or perhaps half hidden behind the faucial pillars, or it may be seen in discrete patches before these have coalesced. In ordinary follicular tonsillitis the exudate is more yellow and can be easily wiped away, and the formation of membrane is exceptional. Membrane may, however, form in Vincent's angina, but the bacteriological findings will distinguish this condition. A peritonsillar abscess may be distinguished by the absence of fœtor, the smaller amount of glandular swelling, and the difficulty that the child has in opening the mouth. The rare condition of agranulocytic angina, characterised by gangrenous inflammation of the throat and disappearance of polymorphonuclear cells from the circulation, is distinguished by the remarkable blood picture.

The diagnosis of diphtheritic laryngitis is made easier if there is any visible membrane in the throat, but often there is no membrane to be seen. The presence of an increasing degree of stridor and breathlessness is however suggestive, and antitoxin should be given pending the examination of a throat swab, for there is no variety of diphtheria which demands more prompt administration of serum. There may be difficulty in young children in distinguishing laryngeal diphtheria from a retropharyngeal abscess, but when the latter is present there is usually a visible bulging of the posterior pharyngeal wall, or the abscess may be felt by direct palpation.

A blood-stained nasal discharge with excoriation of the skin always suggests diphtheria, but when the discharge is unilateral a foreign body in the nose must be excluded by the history and local examination.

Bacteriological Diagnosis. A throat swab should be taken in all acute exudative inflammations of the throat, for diphtheria bacilli may sometimes be found even when the clinical appearances do not suggest the disease. A swab should also be taken in cases which are clinically diphtheritic, in order to confirm the diagnosis. Occasionally swabs from suspected carriers and from children with nasal discharge show the presence of organisms which morphologically resemble diphtheria bacilli. If there is any doubt as to the significance of these organisms, their virulence should be tested by guinea-pig inoculations.

Generally eighteen to twenty-four hours must elapse after a throat swab is taken before the bacteriological diagnosis of diphtheria is established, and meanwhile it may be courting disaster to withhold antitoxin serum from a child who is under suspicion. The importance of giving serum at once to any child in whom there is good reason to suspect diphtheria cannot be too strongly emphasised.

Prognosis. The principal factor affecting prognosis is the interval which elapses between the onset of the infection and the administration of antitoxin serum. The mortality is negligible if serum is given during the first forty-eight hours, but then rises steadily with each day that serum is delayed. Age also affects the prognosis, the younger the child the worse being the outlook. When toxic symptoms have already developed or when signs of post-diphtheritic paralysis have appeared, serum has no influence and the prognosis is accordingly grave. The mortality is high in laryngeal diphtheria, although fortunately the use of serum has largely diminished the incidence of this variety.

Immunity after one attack of diphtheria is not permanent. Second attacks occasionally occur at an interval of a few years after the first attack.

Treatment. *Diphtheria Antitoxin.* Antitoxic serum is measured in units, a unit being the amount required to neutralise 100 minimal lethal doses of toxin for a guinea-pig of standard weight. The amount of serum to be given is determined by the extent and duration of the disease, not by the age of the patient. In a doubtful or mild case an initial dose of 4,000 units may be given while the diagnosis is being established. If the diagnosis is undoubted but there are as yet no signs of toxæmia 15,000 units should be given. When evidence of toxæmia is already present, higher doses of from 30,000 to 60,000 units are required, but it must be remembered that the value of serum depends on its being given early, and an increased dosage will not compensate for delay in using it. A concentrated serum (B. W. & Co.) containing 1,500 units per c.c. has obvious advantages for the patient. A second injection of half the initial dose should be given in twelve to twenty-four hours, and in severe cases this dose may need to be repeated daily for three or four days. The serum should be given intramuscularly, the vastus externus muscle on the outer side of the thigh being the best site because the comfort of the patient lying in bed is but little interfered with.

In the most severe cases the serum may be given intravenously.

The injection of serum induces a state of hypersensitivity to later doses, but this does not develop for ten days, and until then injections may be repeated with safety. If they are given after this period they may be followed by alarming symptoms of anaphylaxis such as rigors, sudden pallor, or syncope. In that event an immediate injection of adrenalin should be given, but the likelihood of anaphylaxis may be avoided by first inquiring whether the child has ever been given serum previously. If this be the case a preliminary test for sensitivity should be carried out by injecting one or two minims of serum intradermally; if this does not cause any reaction the larger amount can then be given intramuscularly.

Serum Sickness. Not infrequently an injection of serum is followed after an interval of about ten days by the appearance of an urticarial or erythematous rash, with pains in the joints and occasionally acute abdominal pain. The temperature is raised and there is often vomiting. The rash first develops round the site of injection and from there may rapidly spread over the whole body. It continues to appear for two or three days and often causes intense itching.

In order to prevent the rash of serum sickness Rolleston recommends giving 5 grains of calcium chloride thrice daily. When the rash has appeared an ointment containing one drachm of menthol to the ounce of soft paraffin may be used. Aspirin in 5- or 10-grain doses three times a day will help to relieve the joint pains.

General Treatment. The child must, of course, be strictly isolated, and should be nursed lying flat in bed with only one small pillow. The possibility of sudden cardiac paralysis makes it imperative that the child should not be allowed to sit up at all nor do anything for himself. In the acute stage the diet must be light, and will consist chiefly of milk or thickened milk foods. Simple fluids should be given in plenty, and if there is frequent vomiting saline should be given rectally. The bowels should be regulated by enemata in order to avoid any straining.

Provided that no signs of cardiac dilatation or paralysis have appeared, another pillow can be allowed after the third week, and when a month has elapsed the child can sit up in bed, and may get up after six weeks. As each advance is made in convalescence a close watch should be kept on the pulse rate for the appearance of tachycardia, for a rapid or irregular cardiac action will call for a further period of rest. Local treatment of the throat has become of little consequence since the use of serum, and indeed the amount of disturbance caused by painting the throat or trying to remove the membrane does much more harm than good. For older children who are prepared to acquiesce in having their throats sprayed, a weak solution of chloramine-T may be used.

Treatment of Diphtheritic Laryngitis. This depends on the severity of the symptoms. Big doses of serum (30,000 to 60,000 units) will be required. Inhalations of steam may help to ease the breathing, and a dose of bromide by mouth or by rectum will help to soothe the child and quieten the respirations.

Tracheotomy [1] is indicated when there is persistent dyspnœa and restlessness, or if after a few hours it becomes apparent that stridor, cyanosis, or recession of the chest wall are increasing. If there is any doubt about the need for tracheotomy it is better to proceed with the operation, for delay may prove fatal, and is certainly likely to make a later operation a hurried and difficult affair. Intubation, which consists of passing a tube from the mouth through the larynx, is a matter for experts, and it is generally better to employ tracheotomy.

Prevention. The prevention of diphtheria has made considerable strides in the last few years since the introduction of the Schick test, which indicates whether a person is susceptible to or is immune from the disease. The test is performed by injecting intradermally into the forearm 0·2 c.c. of diluted diphtheria toxin, while into the opposite arm is injected as a control the same amount of diluted toxin which has been inactivated by heating. The reaction can be read after twenty-four to forty-eight hours, although a later reading after five days or so is necessary in order to detect late reactors. A positive reaction consists of a raised, red, œdematous area ½ to 1 inch in diameter. The reaction reaches a maximum in from three to five days and then slowly subsides, leaving a brownish stain in the skin. A practical difficulty in reading the test is that pseudo-positive reactions may be caused by the proteins in the solution used for testing, but a reaction of this sort affects the control as well as the arm used for the test. A pseudo-positive reaction reaches its maximum in about thirty-six hours and consists of a raised red œdematous area, which is, however, less extensive than a true positive and also disappears more quickly.

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[1] *The Operation of Tracheotomy* (with acknowledgements to "Diseases of Infancy and Childhood," edited by Parsons and Barling, 1933).

It is essential to define and fix the cricoid cartilage which is then held between the thumb and second finger of the left hand, with the tip of the index-finger coinciding with the lower edge of the cartilage. These fingers must not move until the dilators are in place. Then starting from the tip of the index-finger, a tentative incision half an inch or less in length is made absolutely in the midline. The cut is deepened until the level of the trachea is reached. Next, with the knife held nearly vertical, a cut is made through the trachea of the same length as the skin incision. As soon as the trachea is opened, air escapes with an unmistakable sound. Guided by the tip of the left index-finger, which is still in position, the spring dilating forceps are then introduced and the blades opened slightly, *not* fully. A violent expiration with the expulsion of membrane usually follows. When the airway is clear and the dilators are in position, the fingers may be removed from the cricoid. If respiration through the tracheal wound is quiet and regular, the tracheotomy tube selected, minus its inner tube, is inserted between the blades of the dilators either with the pilot provided or better with the fingers. If the airway is still satisfactory and the child comfortable—it is quite likely to have fallen asleep—the tapes are tied round the neck, a gauze dressing inserted between the skin and the cross-piece of the tube, the inner tube inserted and locked, and the child returned to bed.

Subsequently, the inner tube must be removed at intervals for cleansing. As a rule the tube may be removed entirely in from two to four days after the operation and the wound allowed to granulate.

If a clear airway is not obtained when the dilators are inserted, an endeavour must be made to remove any occluding membrane with the membrane forceps inserted between the dilators. To this end it may be necessary to continue the incision downwards. The two main accidents of tracheotomy are (i) a lateral incision which may occasion severe venous hæmorrhage, and (ii) an incision too high involving the cricoid. This may result in stenosis.

Because of the pseudo-positive reactions there are four possible responses to the Schick test, and these are set out in the following table :—

At site of test injection.	At site of control injection.	Interpretation.
1. True positive . .	Negative	Person susceptible
2. True positive and pseudo-positive .	Pseudo-positive	Person susceptible
3. Pseudo-positive .	Pseudo-positive	Person non-susceptible
4. Negative . .	Negative	Person non-susceptible

The Schick test varies with the age of the child. Infants have a natural immunity to diphtheria, and the Schick test in them is negative, but by the end of the first year this immunity has been lost, and the majority of children from then until six years of age are susceptible to the disease. In the later years of childhood the number of positive reactions falls off owing to immunity having been gained either by a definite attack of diphtheria or through exposure to mild and unrecognised doses of infection.

Immunisation. The prevention of diphtheria consists of creating an active immunity by the injection of small doses of diphtheria toxin, or to be more exact, by a mixture of toxin which has been treated with formalin (toxoid), and antitoxin. The two preparations commonly employed are toxin-antitoxin floccules (T.A.F.) and alum precipitated toxoid (A.P.T.). Of the former, three intramuscular injections of 1 c.c. each are given at weekly or fortnightly intervals ; alum precipitated toxoid has the advantage that only two injections of 0·1 c.c. and 0·5 c.c. are required, but it is more likely to cause local reactions than is T.A.F. The acquisition of immunity is tested six weeks after the injections by performing the Schick test. This will be found negative in from 80 to 100 per cent. of the cases. It is also usual to carry out a Schick test before the injections are given in order to determine whether the child is already immune or not, but this may be omitted under the age of six years or so, as the great majority of children are at that period positive.

Active immunisation against diphtheria is being carried out on a steadily increasing scale, because wherever it has been employed there has been a notable diminution in the incidence of the disease.[1] It must be understood that immunity does not prevent a child from harbouring virulent organisms in the throat and thereby acting as a carrier. Once active immunity has been produced it persists for a number of years, but even though a child may have been immunised, if a sore throat develops which has the clinical appearance of diphtheria, the usual treatment should be applied. The proven value of the Schick test and the tremendous advantage to the child population of active immunisation will not be shaken by the rare occurrence of diphtheria in a previously immunised child.

Treatment of a Diphtheria Carrier. The treatment of the child who, though healthy, carries virulent organisms in the nose or throat is important, although, from a public health standpoint, diphtheria could never be controlled by attempting to eradicate carriers, for they are so numerous. The organisms should first be tested for their virulence in order to be sure that they are not actually harmless diphtheroids. When the organisms are obtained from a throat swab it will generally be found that the patient has unhealthy tonsils and adenoids, and the removal of these is usually sufficient to clear up the infection. The treatment of nasal carriers is less satisfactory. Alkaline nasal douches or an antiseptic nasal spray of acriflavine solution (1 : 5,000) may be employed, but there is general agreement that the local application of antiseptics is disappointing.

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[1] As an example, in the City of Manchester between the years 1930–36, the attack rate per 1,000 immunised children under fifteen years of age averaged 0·34 compared with an attack rate of 4·92 in non-immunised children of the same age group. Public recognition of the value of immunisation has been considerably greater abroad than in this country, which may be illustrated by the fact that in 1936 the death rate from diphtheria per 100,000 children under fifteen years of age was only 2·1 in New York, compared with 31·8 in England and Wales.

Scarlet Fever

Scarlet fever and diphtheria are analogous in that they are both due to a local infection of the throat by a specific organism, followed by general effects due to the absorption of toxin. Just as diphtheria is occasionally met with in other situations than the throat, so scarlet fever may sometimes arise from superficial skin lesions or from a surgical wound—so-called surgical scarlet fever.

The investigations of the Drs. Dick in 1923 established the "streptococcus scarlatinæ"—a member of the family of hæmolytic streptococci—as the causal organism, and it is now generally recognised that hæmolytic streptococcal tonsillitis and scarlet fever are essentially identical conditions. The rash of scarlet fever is caused by a rash-producing or erythrogenic exotoxin from the streptococcus; and in order for a rash to appear, not only must the infecting streptococcus have the power to produce this toxin, but the patient must be susceptible to it. Should the patient be immune to the toxin, a rash will not develop, and the diagnosis of scarlet fever will not be made, but the infection may pass from this patient to another who happens to be susceptible to the toxin, and then the rash of scarlet fever will appear. The risk of complications—otitis media, cervical adenitis, nephritis, carditis—is the same whether or not a rash has appeared: and a patient with hæmolytic streptococcal tonsillitis, but without a rash, is just as infectious as the one with a rash, and should be as carefully isolated.

Etiology. Scarlet fever is principally a disease of temperate climates, and in this country has its greatest incidence in the autumn and winter months. It is rarely met with in infancy, but from then on its incidence rises rapidly to reach a climax between five and ten years of age. The sexes are equally affected.

The disease is spread chiefly by droplet infection of sputum from persons harbouring the organism in their throat. Such persons are as a rule suffering from the symptoms of scarlet fever, but it is probable that infection may also take place by carriers, who, themselves immune from the disease, convey the organism in their throat. Discharges from the ear or nose arising during the course of the disease are also a potent source of infection, particularly as these discharges may continue for some time after the other symptoms have subsided. It used to be held that the peelings of the skin in scarlet fever were a source of infection, but the rash and succeeding desquamation are now accepted as the result of streptococcal toxæmia, and the scales are not infectious unless contaminated by the patient's discharges. It has also been shown that fomites, such as books and toys, which have been handled during the patient's illness may convey the organism, but the part played by them is relatively slight. Occasionally outbreaks of scarlet fever have been traced to milk which has been infected by human contact.

The incubation period of scarlet fever is generally two or three days, but may be as long as a week. Contacts should be isolated for ten days.

Symptoms. The onset is sudden, and is characterised by headache, vomiting, and a sore throat. The temperature rises to 102° to 104° F., the pulse is rapid, and the skin is hot and dry. Vomiting almost always occurs at the onset, and is in fact as typical of scarlet fever as it is of acute pneumococcal infections. Older children are likely to complain of soreness of the throat and may refuse their food, but younger children often give no indication that their throat is inflamed, and unless a routine inspection of the throat is made as soon as the fever is discovered the cause of the illness is likely to be overlooked until such time as the rash appears.

Examination of the throat shows it at first intensely reddened and injected. If the tonsils are present they are swollen, and after twelve hours or so patches of yellow purulent exudate appear on them, and the lymphatic glands at the angles of the jaw become enlarged and tender. There is, however, nothing in the appearance of the sore throat of scarlet fever to distinguish it from an ordinary follicular tonsillitis.

The rash develops after twenty-four to forty-eight hours, appearing first on the front of the chest and quickly spreading to cover the whole trunk and the limbs. It is a bright red colour, and consists of closely-set pin points of erythema which can be faded by pressure. The fine follicular character is best seen on the trunk. The rash is generally most severe over the lower abdomen and on the inner aspects of the thighs. The face appears flushed, but the skin round the mouth and nose is often unaffected and appears pale by contrast—circum-oral pallor. At this stage the throat inflammation is at its maximum, and can usually be described as a severe follicular tonsillitis. The tongue is coated with a heavy white fur, and the breath is unpleasant.

The rash begins to fade in from two or three days to a week, and at the same time the throat begins to clear, the temperature falls by lysis, and the tongue loses its furring, becoming the dull red colour of a ripe raspberry.

After the rash has disappeared the skin begins to desquamate; this usually starts at the end of the first week and may go on for a month or six weeks. Desquamation begins on the face and trunk in the form of fine circular scales, and then spreads down the limbs to finish on the palms of the hands and the soles of the feet where the skin may sometimes be shed in large flakes. Occasionally the same area of skin may peel more than once. As in typhoid, the child may shed his hair at the termination of the disease.

Varieties. The description given above is that of scarlatina simplex—the usual form of scarlet fever. In the *anginoid* form the inflammation of the throat is very severe, the face appears swollen owing to lymphatic obstruction from cellulitis in the neck, suppuration of the cervical glands may occur, and there is a risk of death from septicæmia. In the *malignant* form the degree of toxicity is high, the fever may be hyperpyrexial, the pulse is rapid and feeble, respirations are quick and shallow, vomiting may become persistent, and within a day or two the child dies in coma. In these cases the rash may be purpuric instead of erythematous, but fortunately such severe instances are rarely met with at the present day.

Complications. Complications may be due to a local spread of infection from the throat, and include such conditions as suppurative cervical adenitis and acute otitis media. The latter may progress to mastoiditis and the various intracranial complications of middle ear disease. A spread of infection downwards into the chest, causing bronchitis and broncho-pneumonia, is fortunately uncommon.

At the end of the first week, and during the second and third weeks, complications may arise in more remote parts of the body, and are probably due to the effects of toxic absorption. The two most important are acute nephritis and acute rheumatism. Acute nephritis is as likely to follow a mild as a severe attack, puffiness of the eyes and of the face being as a rule the first warning that the kidneys are involved. Vomiting is usual at the beginning of the nephritis, but it does not continue for long unless the kidneys are severely damaged, and is then an indication of uræmia. The urine is strongly acid, reduced in amount and with a raised specific gravity, and usually contains enough blood to give it a red or smoky appearance. There is a heavy albuminuria, and microscopical examination shows blood cells, a few granular casts, and an occasional blood cast.

As a rule the nephritis recovers completely. The most severe cases are likely to be those in which the onset of the nephritis has been overlooked, often because the proper significance of the original throat infection has not been recognised, and the child has been allowed to be up and about before the nephritis begins. The course of the illness is then often prolonged, and may gradually merge into the chronic œdematous type of kidney disease. It has been suggested that the occurrence of nephritis can be largely prevented by giving big doses of alkalis during the acute stage of the fever ; for this purpose 15 gr. each of potassium citrate and sodium bicarbonate may be prescribed three times a day.

The rheumatism which follows scarlet fever is indistinguishable from rheumatic fever. As a rule the symptoms are those of multiple acute non-suppurative arthritis, but rheumatic carditis may occur as well. The rheumatic symptoms generally appear at the end of the first week, and the attack is usually the first that the child has experienced, although if there have been previous attacks of acute rheumatism the occurrence of scarlet fever is very likely to provoke a relapse. Rapid disappearance of the arthritic symptoms under treatment with salicylates is an additional point in favour of identifying the rheumatism of scarlet fever with ordinary rheumatic fever. If the heart is involved it must be treated on the usual lines for rheumatic carditis.

Quite distinct from the rheumatic arthritis is the occasional occurrence of suppurative arthritis after scarlet fever, due to the presence of pyogenic organisms in a joint. In a different class, too, are the muscular cramps and fleeting pains in the limbs which often occur after the children have been allowed to get up : these are due to muscular fatigue, often associated with some degree of flat feet, but are not truly rheumatic.

Diagnosis. Diagnosis must be deferred until the typical eruption appears. The yellow follicular character of the exudate in the throat is unlike the appearance seen in diphtheria, but it is a wise precaution to swab the throat in order to exclude the latter infection. The culture of hæmolytic streptococci on the swab does not necessarily imply that the child will develop the rash of scarlet fever, for the organism may be some other member of the hæmolytic streptococcal family, or possibly the child may be already immune to the rash-producing toxin.

The appearance of the rash, taken in conjunction with the sore throat, generally puts the diagnosis beyond doubt, but it must be remembered that similar rashes may be produced by such drugs as belladonna, salicylates, chloral, and quinine, and may occasionally follow a soap and water enema. The rash of German measles may sometimes resemble scarlet fever, but the milder fever, the slight degree, if any, of inflammation of the throat, and the enlargement of the occipital glands are points of distinction. In recent years the Schultz-Charlton blanching test has been used in helping to decide whether a rash is scarlatinal. 0·2 c.c. of a 1 : 10 dilution of scarlatinal antitoxin is injected intradermally into the area of the rash ; if it is scarlatinal the antitoxin causes it to fade over an area of an inch or two, and the blanching persists for a day or so. Unfortunately the test is most distinct, and therefore of greatest use, when the rash is already so severe as to leave little doubt of the diagnosis.

Prognosis. The virulence of scarlet fever has been declining for some years. In the last fifty years the mortality has dropped from 13·5 per cent. to less than 1 per cent. What little mortality there is is due largely to septic complications, and the recent introduction of anti-scarlatinal serum has still further improved the outlook by lessening the incidence of these. One attack of scarlatina does not necessarily confer a lasting immunity, for second attacks may occur months or years later. Relapses are occasionally met with a few weeks after the initial attack.

Treatment. The child must be nursed in bed and be strictly isolated. In a private house he must have a room to himself, using his own set of feeding utensils, and his clothing and bedding must be sterilised and washed separately. So long as the temperature is raised the diet should be mainly of fluids, such as barley water, lemonade, fruit drinks, and milk. Confinement to bed should continue for three weeks, in case such complications as rheumatism or nephritis appear. Provided that the child is free of discharges from the ear or nose, he may be allowed to mix with other children after four weeks.

The employment of anti-scarlatinal serum has proved of great value. It is an antitoxic serum, not antibacterial, and its effect is most dramatic when toxæmia is severe, for it quickly reduces the temperature, relieves the headache, hastens the disappearance of the rash, and diminishes the likelihood of complications, although if complications have already arisen they are not affected by the serum. It should be used in all severe cases, but in milder cases the disturbance due to the serum may be more unpleasant than the effects of the disease. The serum is given intramuscularly in doses of from 10 to 20 c.c. Generally one dose is sufficient, but in severe cases it may be given daily for two or three days. Being a foreign serum (prepared from horses) its administration is usually followed in about ten days by serum sickness.

The sulphanilamide group of drugs occupies a place in treatment similar to that in non-scarlatinal tonsillitis (see p. 319). Not only do they cut short the inflammation in the throat, but they also diminish the likelihood of those complications such as cervical adenitis, quinsy, and otitis media, which arise as a result of direct spread of infection from the throat. They cannot take the place of anti-scarlatinal serum, since they have no direct influence on the toxæmia, and their use does not prevent the employment of serum in severe cases.

Local treatment may be used to relieve the discomfort in the throat. The child may be given small pieces of ice to suck, and the throat may be sprayed with an antiseptic oil such as eucalyptus oil m. 15 and menthol gr. 5 to the ounce of liquid paraffin, or a solution of potassium permanganate (gr. 15 to the ounce of water) may be used instead. Painting the throat is a useful measure when it can be done without causing much disturbance ; the following paint serves very well :—iodine gr. 6, acetic ether dr. 2, glycerin ad. oz. 1. For children who are old enough to gargle, 10 gr. of potassium chlorate to the ounce of water is suitable, and glycothymoline is also a pleasant preparation. Whether local treatment is to be advised, however, must depend on the behaviour of the child, for if it is resented and gives rise to struggling, it is better omitted.

If there is much pain and stiffness of the neck, warm fomentations may be applied, but these should only be used until the tenderness has been relieved, as otherwise they may promote abscess formation in the cervical glands. Delirium, hyperpyrexia, and restlessness at night are all best dealt with by tepid sponging. A daily action of the bowels must be ensured, and aspirin may be given to relieve the headache.

Prevention. This depends mainly upon prompt isolation of patients infected with the hæmolytic streptococcus. Susceptibility to the rash-producing or erythrogenic toxin can be determined by means of the Dick test, which consists of the intradermal injection of 0·2 c.c. of diluted toxin, a positive reaction appearing as an area of erythema at the site of injection after twenty-four hours. A positive reaction indicates that infection with the streptococcus scarlatinæ will be accompanied by the rash of scarlet fever. The test is likely to be negative during the first year, from then until three years of age it is usually positive, after which the percentage of positives begins to decline. The test is also positive during the first three days of scarlet fever.

Active immunity against the rash-producing toxin may be conferred by injections of small amounts of the toxin, while passive immunity can be given by injections of antitoxin. Either type of immunity does not prevent infection by the hæmolytic streptococcus, and merely to immunise against some of the toxic effects of such infection has proved disappointing, and for the most part has been abandoned.

Measles (Morbilli)

Measles is one of the most infectious of the exanthemata. Although there can be little doubt that the disease is due to a specific organism it has not as yet been isolated. The infection is conveyed principally by discharges from the throat, nose, and eyes, particularly during the first few days of the disease. Fomites are probably of little importance.

Measles is most common between the ages of two and seven years. Infants of a few weeks old are usually immune, but their immunity is soon lost. The disease is most prevalent in the second half of the winter, and assumes epidemic proportions every two or three years. Measles and whooping-cough not uncommonly follow each other within a short interval, measles being more often the sequela to whooping-cough than *vice versâ*. Both are likely to be complicated by broncho-pneumonia, and the occurrence of all three is usually followed by some permanent scarring of the lungs.

The incubation period ranges from seven to twenty-one days, but is usually ten days.

Symptoms. Within a few hours of being exposed to infection the child may develop fever, slight nasal catarrh, conjunctivitis, and even a fleeting rash, symptoms which have been termed the "illness of infection." They only last for a day or so. The illness proper, which comes on at the end of ten days, has two stages. There is at first a prodromal period lasting three or four days and characterised by fever and catarrhal symptoms. This is followed by the appearance of the typical eruption which lasts for another three or four days.

The onset of the prodromal period is accompanied by headache, loss of appetite, and a temperature up to about 101° F. Vomiting is usual. Catarrhal symptoms rapidly develop, the conjunctivæ become infected, the eyes are watery or they may discharge a thin purulent fluid, and there is a complaint of photophobia. There is also a copious muco-purulent discharge from the nose, and epistaxis is likely to occur. The throat is infected, and the voice may become hoarse or feeble owing to laryngitis. At times the laryngeal symptoms are very prominent and may even cause some confusion with laryngeal diphtheria. The watery appearance of the eyes, the stuffy and discharging nose, and the miserable appearance of the child form a picture which at once suggests the true nature of the illness.

Examination of the chest in the prodromal stage usually shows signs of bronchitis, and in the most severe cases this may rapidly progress to broncho-pneumonia. In infants this stage is usually accompanied by diarrhœa. In about half the cases the catarrhal symptoms are accompanied by prodromal rashes, which may consist of a blotchy erythema or may be urticarial or scarlatiniform. They tend to be more localised than the characteristic eruption which is to appear later on, and are also more transient, lasting for only a day or so.

The diagnosis of measles during the catarrhal stage is confirmed by the appearance of *Koplik's spots* in the mouth. They were originally described by Koplik in 1896, and occur in something over 80 per cent. of cases, and their presence puts the diagnosis beyond doubt. To see them the child must be examined in a good light, preferably daylight. The spots appear as bluish-white pin points, each surrounded by a narrow ring of bright red injection. Their usual situation is on the mucosa of the cheeks on one or both sides, close to the lower alveolar margin opposite the premolar or molar teeth. They vary considerably in number ; as a rule there are about four or six, but occasionally they are so numerous as to smother the inside of the cheeks. Koplik's spots precede the characteristic eruption by two or three days, and they last for four or five days, but always disappear before the subsidence of the rash.

The typical rash of measles develops on the fourth day of the illness, and is accompanied by a recrudescence of fever to 103° F. or so, and by some increase in the signs of catarrh. The rash appears first behind the ears, and spreads rapidly to cover the face, trunk, and limbs. The eruption consists at first of small red macules, which soon enlarge and coalesce to form blotchy papules. These are irregular in shape, but the crescentic outline which is often described can seldom be made out. The rash is most intense after forty-eight hours, and is often best seen on the face ; it then begins to fade, retreating in the same order as it appeared, and in an uncomplicated case the temperature now returns to normal, and the catarrhal symptoms clear up. A slight brown staining of the skin may remain for a week or two. During the eruptive stage leucopenia is usual.

Varieties. Varieties of measles are uncommon. There is an abortive form in which the rash is poorly developed and makes but a fleeting appearance, but the presence of Koplik's spots is sufficient to confirm the diagnosis. A suffocative form is occasionally met with in epidemics in overcrowded institutions, and is likely to be fatal. The appearance of bullæ with the rash is also a rare but fatal variety.

Complications. The most common complications are those that affect the respiratory tract. During the early catarrhal stage bronchitis is usual, and may quickly spread to the lung alveoli giving rise to broncho-pneumonia. This is most likely to happen in young children, and is always a most dangerous complication. Recovery from the broncho-pneumonia is likely to take a long time, and resolution of the lung is often incomplete, leading to a variable degree of permanent pulmonary fibrosis. In later childhood this may account for recurring attacks of bronchitis each winter, or may lead to more definite evidence of bronchiectasis. There is also no doubt that the injury to the lungs in measles makes them more prone to tuberculous infection, or, should a tuberculous focus already be present, it is very likely to light up and spread during the months that follow.

Of other complications otitis media is common. Laryngitis, which is frequently present in the early stage of the illness, may be sufficiently severe to cause obstructive dyspnœa, and may occasionally go on to deep ulceration. Similarly the inflammation of the conjunctivæ, which is usual during the early catarrhal stage, may spread to involve the cornea, and lead to permanent opacities ; indeed, it occasionally happens that all the structures of the eye become inflamed—panophthalmitis—leading to complete blindness. The catarrhal stomatitis may sometimes progress to ulceration, which as a rule heals readily under local applications. Gangrene of the mouth is fortunately rare.

Encephalitis is an occasional complication. The symptoms appear at about the time when the rash is fading, and consist of increasing drowsiness or coma, accompanied by some stiffness of the neck, perhaps convulsions, and sometimes local paralyses. The mortality is about 10 per cent. When recovery takes place it is usually complete, without nervous or mental sequelæ.

Diagnosis. Measles may easily be overlooked until the fourth day, when the typical rash is due to appear, for during this period the symptoms resemble those of a severe cold, or they may be confused with tonsillitis or simple laryngitis. The fleeting rashes which may appear during the prodromal period may be mistaken for the eruptions of other specific fevers, or even with the later rash of measles.

In infancy the onset may be marked by such acute diarrhœa and vomiting that suspicion of measles is not entertained, and this is of course particularly unfortunate if the infant should be admitted to a ward with other children, for the infectivity of measles is probably greatest during the early catarrhal stage. The same may be said of those cases, usually in young children, in which broncho-pneumonia is an early complication, for the very severity of the lung condition may make one overlook the possibility of measles, especially as these are just the cases in which the rash may be slight or "suppressed." The finding of Koplik's spots during the catarrhal stage is however a most reliable diagnostic criterion, and the examination for these tell-tale spots should therefore never be omitted.

When the rash appears it may be mistaken for German measles, but in the latter condition the relatively slight disturbance of health, the absence of Koplik's spots, or of preceding catarrh, and the usual enlargement of the cervical and occipital glands will serve as distinguishing features. Occasionally drug rashes, and the rashes of serum sickness are morbilliform, but the associated symptoms of measles are lacking.

Prognosis. The mortality varies considerably in different epidemics. The disease is most likely to be fatal during the first two years of life, the mortality at that age approaching 20 per cent. Death is usually the result of broncho-pneumonia, and in young children any condition which promotes respiratory infection, such as malnutrition, rickets, or previous whooping-cough, affects the outlook adversely. Diarrhœa, or the various complications of otitis media, account for other fatal cases. Relapses and second attacks of measles are very rare.

Treatment. Strict isolation is, of course, necessary. The child should be nursed in bed in a warm, airy room, and so long as the temperature is raised the diet must be mainly fluid—milk, milk puddings, custard, junket, broths and jellies. In order to protect the eyes the room should be darkened, and reading should not be allowed. The conjunctivæ should be bathed morning and evening with a weak boracic solution. The mouth and throat must also be kept as clean as possible by the use of mouth washes and throat sprays, and older children should gargle with glycothymoline or a solution of potassium chlorate (gr. 12 to the ounce of water). Laryngitis is best treated by keeping the air moist with a steam kettle, the steam being impregnated with Friar's Balsam (2 teaspoonfuls to the pint of water). Exceptionally the dyspnœa may be severe enough to require tracheotomy. It should be remembered that diphtheria and measles may sometimes co-exist, and when laryngeal symptoms are severe, and the question of diphtheria has been raised, it would be wise to give antitoxin at once before waiting for a bacteriological report. The management of broncho-pneumonia should follow the usual lines (see p. 360).

In the absence of complications, the child can be allowed up and may mix with others a fortnight after the onset of the illness. Bearing in mind the considerable degree of debility which often follows measles, a period of convalescence should be arranged in the country or at the seaside for two months or so, and should be combined with suitable tonics such as Parrish's Food or cod-liver oil and malt.

Prevention. The serum of a patient convalescent from measles contains immune bodies, and during the last few years success has attended attempts to protect children who have been exposed to the disease by injecting into them serum obtained from convalescents. The immunity afforded by this means is of course passive, and only lasts about a month.

If serum is injected during the first six days after exposure to infection, complete protection may be expected ; if the injection is delayed to between the sixth and ninth day, the attack of measles will be modified, but if it is delayed until the prodromal symptoms have appeared, the course of the illness is not affected, although the rash does not appear at the site of injection (Debré's phenomenon). In dealing with an outbreak in a ward the serum should be given to all contacts as soon as possible after exposure to infection in order to prevent the disease from spreading, but for the child in his own home there is an advantage in delaying the serum until the seventh day, for then he will have a very mild attack which will, however, be sufficient to confer a permanent immunity. The serum is injected intramuscularly, a suitable dose being 3 c.c. up to three years of age, and an additional c.c. for every succeeding year. The serum being human, there is no risk of anaphylaxis or serum sickness.

If convalescent serum cannot be obtained, serum can be prepared from an adult who has previously had measles, but double the dose needs to be given, and even then the protection is less reliable. It has also been shown that globulin extracted from the placenta contains immune bodies against measles, and can be used prophylactically. Two c.c. are given intramuscularly, and the injection is repeated in two days. General and local reactions may follow. The degree of protection is equivalent to that afforded by adult serum, which modifies, without entirely preventing, an attack.

German Measles (Rubella) (Roseola)

This is an acute specific disease, mildly infectious, characterised by a rash which may either be morbilliform or scarlatiniform, and associated with an enlargement of cervical and occipital lymph glands, but giving rise only to slight constitutional disturbance.

Rubella has a world-wide distribution, and in this country occurs principally in the spring. It is rare in infancy, and most commonly affects children between five years of age and puberty. The disease is spread by the patients themselves, carriers and fomites not being of importance. The causal organism is not known.

Symptoms. The incubation period varies from one to three weeks, but is generally between fourteen and seventeen days. As a rule the first symptom is the rash, but it may be preceded for a day or so by slight fever, headache, and pharyngitis. The rash appears first on the face, and spreads to the trunk and limbs. It usually consists of closely arranged erythematous spots which vary in size from a pin's head to a haricot bean. These may coalesce to some extent, and the rash then closely resembles that of measles, while at times the eruption becomes confluent and simulates that of scarlet fever. Both morbilliform and scarlatiniform types may be present together. While the rash is out the temperature is raised to 100° F. or so, the throat is injected, and the conjunctivæ may be a little congested, but these symptoms are slight and in two or three days when the rash fades they quickly disappear.

Enlargement of the occipital and cervical glands can generally be made out, and helps to confirm the diagnosis. The glands become palpable just before the rash appears, and they may not subside for a week or more after the rash has faded. They are a little tender and may make the neck stiff, but they do not go on to suppuration.

Diagnosis. The two diseases that are likely to be confused with rubella are measles and scarlet fever, but in both of them the amount of constitutional disturbance is a good deal greater. The prodromal catarrh and Koplik's spots will distinguish measles ; in scarlet fever the throat is more severely inflamed, the temperature is higher, the rash is more vivid, and the tongue more heavily coated. If doubt still remains, the Schultz-Charlton blanching test for scarlet fever may be employed.

Prognosis. Rubella is the mildest of all the acute specific fevers, and recovery is invariable. Arthritis has been recorded as a complication. Both relapses and second attacks are very rare.

Treatment. The disease is so mild that treatment can be summed up by isolation, rest in bed until the rash has subsided, and attention to ordinary nursing details. The child should remain isolated for a week after the appearance of the rash.

Whooping-cough (Pertussis)

Whooping-cough is essentially a disease of childhood. The youngest instance observed by the author was in an infant aged five weeks, whose mother developed the disease shortly after parturition. It reaches its highest incidence between two and five years of age, and in this country the greatest number of cases occur towards the end of the winter. Girls are said to be affected more frequently than boys.

A small bacillus first isolated by Bordet and Gengou in 1906 is now accepted as the causal organism. It can be recovered from the respiratory secretions during the early catarrhal stage, when the disease is most infectious, but later on is overgrown by other organisms. The disease has been experimentally produced in volunteers by instilling a culture of the organism into the nose and throat.[1] It is spread from one patient to another by droplet infection in the act of coughing and sneezing.

The incubation period varies from one to three weeks, and is generally about 14 days.

Symptoms. The illness begins like an ordinary cold, with sneezing, coughing, nasal catarrh and slight fever. After two or three days the cough is noticed to occur definitely in bouts separated by an hour or more, while between times the child is free of cough. Towards the end of the first week the whoop makes its appearance, and the disease then enters upon its paroxysmal stage.

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[1] McDonald, H. and E. J., *Jour. Infect. Dis.*, 1933, 53, 328.

The characteristic paroxysm consists of a rapid series of expiratory coughs, the child becoming more and more cyanosed, and in severe attacks the face may become bloated, the tongue is projected, the eyes roll up, and the child may seem at the point of exhaustion. The attack finishes by the child making an inspiration through a glottis which is still partially closed, and this accounts for the long crowing or whooping sound. At the end of the paroxysm a small amount of thick tenacious mucus may drool from the mouth, and frequently the attack finishes with vomiting. Each paroxysm may be clearly separated from the next, or four or five may occur in rapid succession. The total number varies widely ; in a mild case there may be no more than half a dozen a day, in a severe case this number may occur every hour. A severe attack is a frightening thing, and children often seem to sense when one is about to come on, and may run to an adult for protection. In the absence of complications the temperature may be normal, or raised only a degree or two, and although the pulse rate and respiration rate are increased with each paroxysm, they quickly return to normal, in fact the child will usually go back to its toys and continue playing until the next burst of coughing.

Physical examination in a simple case may show only a few coarse rhonchi scattered over the lungs ; in more severe cases in which paroxysms are being quickly repeated, the strain on the right side of the heart is considerable, and examination may show a quickened pulse rate, with some degree of cardiac dilatation, and a loud second sound in the pulmonary area. In severe cases the persistent cyanotic tinge and the swollen features give an appearance which at once suggests the diagnosis. A sign which should always be looked for is an ulcer on the frænum of the tongue. It only occurs in about a third of the cases, but when present is diagnostic of whooping-cough. It appears as a pearly white ragged ulcer about as large as a split pea, and is caused by the fretting of the tongue over the incisor teeth during the paroxysms, and therefore is not to be expected before the first dentition. Another characteristic finding is a high lymphocytosis. The white cells generally run up to about 25,000 per c.mm, but have been recorded above 100,000, the lymphocytes accounting for 70 to 80 per cent. of the total cells. The lymphocytosis develops during the early catarrhal stage and persists in the paroxysmal stage, and use may be made of it in the diagnosis of a doubtful case.

Uncomplicated whooping-cough terminates so gradually that it is difficult to set a time to the end of the disease. The paroxysms gradually become less severe and less frequent over a period varying from three to six weeks, and during this time the cough becomes looser and the sputum less viscid. As the paroxysms diminish in severity the child ceases to be worried by them, the appetite improves, and the weight rises. A cough with paroxysmal characters seems in some children to become habitual after whooping-cough, and may continue to be heard at intervals for as long as six months, or may reappear after a year or more if the child should happen to develop a simple cold.

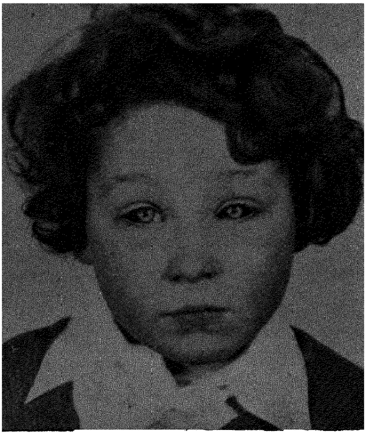

FIG. 132. Subconjunctival hæmorrhages in whooping-cough. Girl aged five years.

Varieties. An abortive form of whooping-cough occurs, in which, after the preliminary stage, the paroxysms are slight and infrequent, and there may be neither a whoop nor vomiting. Such cases are easily overlooked, which is unfortunate because in spite of their mildness they are none the less infectious. It should also be remembered that the whoop may disappear in the face of severe broncho-pneumonia, and that if this should develop during the catarrhal stage the whoop may never be heard. Occasionally attacks of paroxysmal sneezing or hiccough take the place of the cough.

Complications. The violence of the bouts of coughing and the engorgement of the veins during them account for some of the complications; secondary infections, chiefly streptococcal, a count for others. Hæmorrhages are not infrequent, the most common being epistaxis, and hæmorrhages into the conjunctivæ and eyelids may also occur. Intracranial hæmorrhage is fortunately rare ; it is usually meningeal, and is likely to be accompanied by convulsions or coma, and if the child recovers there may be subsequent paralysis, but a fatal termination at the time of the hæmorrhage is more likely. Convulsive attacks accompanying paroxysms of coughing are more frequent in infancy. The violence of the cough may also give rise to interstitial emphysema of the lungs, and pneumothorax has been recorded. Some degree of vesicular emphysema can usually be detected in all but the mildest cases.

The most frequent and important complication is broncho-pneumonia. It is particularly likely to occur in young children, and usually comes on during the paroxysmal stage. The usual physical signs are seldom well developed, and there may be little more than scattered patches of fine crackling crepitations, but the hurried breathing and high pulse rate, together with persistent cyanosis, fever, and a lessening of the whoop all point to this complication. If recovery from the broncho-pneumonia takes place it is a tedious affair, and leaves behind a variable amount of scarring of the lungs.

For months after whooping-cough the child often remains in a debilitated condition, failing to gain weight, and with a poor appetite. Physical examination may often suggest the presence of enlarged mediastinal glands, which may be confirmed by an X-ray examination There will usually be a lurking suspicion that these glands are tuberculous, although it must be remembered that when the resolution of a broncho-pneumonia is delayed the bronchial glands remain swollen for a long time. There is no doubt that whooping-cough, like measles, leaves the lungs more ready to receive tuberculous infection, or if a tuberculous focus was present beforehand and was dormant at the time of the whooping-cough, it is likely to flare up during the next few months. Careful X-ray examination, tuberculin tests, and a search for tubercle bacilli in the sputum or in the gastric washings are of some assistance in trying to decide whether tuberculosis is present or not, but in many cases the question has to remain open. What is quite certain is that a period of some months spent at the seaside will do more than anything else to return these children to health.

Diagnosis. The diagnosis of whooping-cough during the catarrhal stage, that is to say before the whoop has appeared, can hardly be more than a surmise, but even in this stage the cough may show a tendency to paroxysm, with long intervals between each bout. The presence of definite lymphocytosis at this stage would point strongly to whooping-cough. Once the paroxysmal stage has been reached and the whoop has been heard, the diagnosis is simple. Even if the whoop has not actually been heard, the presence of an ulcer on the frænum of the tongue is sufficient to establish the diagnosis. Reliance may also be put upon a bacteriological diagnosis if the method advocated by Gardner and Leslie is adopted. A specially prepared blood-agar plate is held a few inches from the child's mouth during a fit of coughing. In children with whooping-cough the bacillus pertussis can be recovered in this way in about 80 per cent. of cases during the first three weeks of the disease. This affords a means of confirming the diagnosis during the early catarrhal stage, at a time when the disease is most infectious, and when the clinical picture is usually uncertain.

Prognosis. The prognosis depends largely upon the age of the patient. Complications such as broncho-pneumonia, diarrhœa, and convulsions are more frequent in young children, and therefore the younger the child the worse is the outlook. Of 44,000 deaths from whooping-cough in England and Wales 97 per cent. were children under five years of age. The majority of the fatalities occur during the first two years. In older childhood recovery is the rule. The remote prognosis needs to be guarded on account of the delayed convalescence and the possibility of any latent focus of tuberculosis becoming active. Second attacks of whooping-cough are very rare.

Treatment. During the early catarrhal stage, and so long as the temperature is raised, the child should be nursed in bed in a well-ventilated room. When the paroxysmal stage has been reached treatment in the fresh air is of great value, and in the absence of complications and when the temperature is normal, the child should be allowed up on a balcony or, if the weather permits, in the garden, provided that he is kept from contact with other children. Isolation should be maintained for six weeks.

The diet needs to be as nourishing as possible, but dry crumbly foods like biscuits or toast are likely to irritate the throat and start a burst of coughing, and therefore milky foods, broths and soups are to be preferred. Feeding is often made difficult on account of vomiting, but fortunately the appetite as a rule remains good, and food may be retained if it is given immediately after a sick attack.

Numerous drugs have been recommended, but their benefit is seldom very striking. In the catarrhal stage a simple expectorant mixture is all that is required; when the paroxysmal stage has been reached a sedative mixture containing bromide or phenazone (gr. 1 to 4) or luminal (gr. $\frac{1}{4}$), in combination with belladonna, is as useful as any. The following is a suitable prescription for a child of one year:—

℞ Potassii bromidi gr. iii.
Tincturæ belladonnæ m. iii.
Glycerini m. x.
Aquam ad ℥i.

The dose of belladonna may be increased with advantage until $\frac{1}{2}$ drachm is being taken in the day, provided that signs of intolerance do not appear. Burning a cresolene lamp in the bedroom at night-time has sometimes seemed beneficial. When the paroxysms are frequent and severe a watch must be kept on the heart, persistent tachycardia or an increasing dilatation calling for strict rest in bed. In infancy the occurrence of diarrhœa should be dealt with by a small dose of castor oil, dilution of the feeds, and the administration of fractional doses of Dover's powder (gr. $\frac{1}{8}$ to $\frac{1}{4}$), if the condition warrants it.

In severe cases ether may be used to cut down the number of paroxysms. Intramuscular injection has been recommended, but this route causes a good deal of pain and should therefore not be used. A better method, because it is painless, is to give thirty minims of ether in $\frac{1}{2}$ oz. of olive oil by rectal injection twice a day.

An extensive trial has been made of vaccines of B. pertussis and of mixtures of this organism with B. influenzæ and pneumococci. It is generally accepted that vaccines are without effect once the paroxysmal stage has been reached, and the majority of observers have been unimpressed with their use even when given in the catarrhal stage. Serum from convalescent patients has also been tried in treatment but has proved disappointing. The use of vaccines and serum as prophylactic measures is mentioned later.

Convalescence from whooping-cough needs to be thorough. A change of air to the country or seaside is invaluable, the child being allowed to run wild for two or three months. This should be combined with the administration of cod-liver oil and malt.

Other treatments, of very doubtful value but from which benefit has sometimes been claimed, include exposures to ultra-violet light and the application of X-rays to the chest.

Prevention. The prevention of a disease such as whooping-cough, which is both prevalent and in young children carries a high death rate, is obviously desirable. Sauer and others have clearly shown that immunity by means of vaccines can be conferred, provided that the vaccine is prepared from H. Pertussis in its smooth cultural phase, and that large doses of the order of 20,000 million organisms per c.c. are injected.[1] Immunity takes up to two months to develop, and therefore prevention should not be delayed until a child has actually been exposed to infection. From a practical point of view, it is an advantage to combine the injections against whooping-cough with those against diphtheria.

Smaller doses of vaccine given during the incubation period have given conflicting results, but are worth a trial. They should be given before the whoop has appeared. Because secondary infection with catarrhal organisms is so common in whooping-cough, a mixed vaccine of pertussis and catarrhal organisms should be used in this stage.

Chicken Pox (Varicella)

Chicken pox is usually a mild, though highly infectious, illness, characterised by a rash consisting of small vesicles which appear in crops and later dry up into scabs. It is chiefly a disease of childhood, and even infants are not immune. There is no special seasonal incidence. The infective agent is a minute virus, which can be obtained from the fluid in the vesicles in the early stage of the eruption, and is agglutinated by the serum of patients recovering from the disease (Amies). Unfortunately the child with chicken pox may be infectious two or three days before the rash appears, and therefore isolation at the onset of the rash does not prevent the disease from spreading to others.

The incubation period lies between eleven and twenty-three days, and is usually about a fortnight.

Symptoms. During the twenty-four hours that precedes the rash there may be slight prodromal symptoms such as a temperature, headache, and a feeling of malaise. The rash first appears on the chest, and spreads from there during the next twenty-four hours to cover the trunk, the face, the scalp, and the proximal parts of the limbs. The eruption begins as small discrete papules which quickly vesiculate. Each vesicle is quite small, only about two or three millimetres in diameter, and is filled with a clear fluid, and may be surrounded by a narrow rim of erythema. Unlike the vesicles of smallpox they show no umbilication, and are unilocular, so that if one is pricked the whole vesicle empties. After twenty-four hours the fluid in them turns yellow, and in two or three days each vesicle dries up into a scab which separates usually without leaving a scar. As however the rash is very irritating, the child is likely to scratch, and in rupturing the vesicles may infect them, in which case a small pitted scar remains permanently. Most children who have had chicken pox show one or two such scars.

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[1] Antigen laboratories issue a vaccine of this strength. For children under two years, three injections of 1 c.c., 1 c.c., and 2 c.c. are given at intervals of one to three weeks, while for older children the doses are 1 c.c., 2 c.c., and 2 c.c. A mixed vaccine suitable for use during the incubation period can be obtained from Antigen Laboratories, or Parke Davis & Co.

The number of vesicles varies considerably, there may be only a mere half a dozen, or there may be literally hundreds. They do not all appear at once, but come out in fresh crops every day or two, so that after a few days their various stages can all be seen in the same child. The rash lasts as a rule for about ten days to a fortnight.

The distribution of the rash is of importance in diagnosis, particularly in distinguishing the condition from smallpox. The lesions are most numerous on the trunk and back, and the face and scalp may also be freely involved, but on the limbs the number of spots progressively diminishes as the rash travels distally. One or more spots often appear on the palate, and the conjunctiva may also be involved. Any area of unhealthy skin, such as an eczematous patch or a napkin rash is likely to show a thicker distribution of them.

The amount of constitutional disturbance is generally very slight, and apart from a few degrees of fever while the vesicles are appearing the chief trouble arises from the itching.

Varieties. Serious forms of chicken pox are very rare. Occasionally the eruption becomes confluent, and large pustules then form which may leave considerable scarring. In the severe but rare hæmorrhagic form bleeding takes place into the vesicles and there may also be hæmorrhage from the mucous membranes. Varicella gangrenosa is a fatal variety in which the spots become confluent and an extensive slough forms beneath each scab.

Evidence is accumulating that there is a relationship between chicken pox and herpes zoster. Contact with either disease may be followed after the correct incubation period by one or the other, and occasionally both conditions have occurred simultaneously in the same patient. The usual history is that the development of herpes in one member of the family is followed after a fortnight or so by the appearance of chicken pox in other members. On the other hand chicken pox does not confer immunity against herpes zoster, and although second attacks of chicken pox are very rare recurrent attacks of herpes zoster are well known to occur.

Diagnosis. The diagnosis of chicken pox seldom presents difficulty ; the most important distinction is from smallpox. Even in the mild form of smallpox which is occurring at present in this country, the prodromal symptoms of intense headache, backache, and fever are more severe than anything met with in chicken pox. The rash of smallpox is also delayed for two or three days, and the heavy distribution on the forehead and distal parts of the limbs is an important distinguishing feature.

Chicken pox may also be mistaken for papular urticaria, especially when the former occurs in a child who has been known to suffer repeatedly from attacks of the latter condition. The clear watery character of each vesicle and the distribution of the rash will help to prevent error. Chicken pox may occur inside the mouth, but this never happens in papular urticaria.

Prognosis. Varicella is almost always a mild disease, running an uncomplicated course. Relapses and second attacks are very rare.

Treatment. The disease is so mild that no treatment is required beyond isolating the child and nursing him in bed. The itching can best be relieved by repeated bathing in warm boracic baths, allowing 1 oz. of boracic acid to each gallon of water. Alkaline baths (½ oz. of sodium bicarbonate to 1 gallon of water) are also soothing. It is essential that the child should be prevented from scratching himself, so that he may not be left with numerous scars ; for this purpose a baby should wear cardboard splints to the arms, and an older child may wear gloves. Should any of the spots become septic, they may be treated with warm fomentations. As the vesicles dry off they should be dusted with zinc and boracic powder.

Isolation should be continued until all the scabs have separated.

Smallpox (Variola)

Smallpox is an acute infectious disease characterised by a sharp onset with fever, headache, and backache, followed in three days by a rash which goes through papular, vesicular, pustular and scabbing stages.

The disease occurs in two forms—a severe form and a mild one. The widespread practice of vaccination during the second half of the last century and the beginning of the present century reduced the incidence of the severe form to negligible numbers, but during the last fifteen years the milder form has appeared, and because vaccination has to a great extent been abandoned, it has been allowed to gain a footing, especially attacking the child population. Both types are highly contagious, and both are prevented by vaccination. The mild type, which has also been called para-smallpox, is probably identical with Amaas (South Africa) and Alastrim (Brazil).

Etiology. Although no age is exempt, in an unvaccinated community children are particularly susceptible. Congenital instances have been recorded, the mother and her newborn child both suffering from the disease. The disease is caused by a filter-passing virus, almost certainly identical with the Paschen bodies of vaccinia (see p. 696). These bodies can be recovered from the skin lesions, and may be carried by fomites. They are also resistant to drying, and in this state can retain their virulence for years.

The incubation period is generally twelve days.

Symptoms. The illness begins with a prodromal stage which lasts three days before the typical rash appears. Considerable constitutional disturbance occurs at the beginning. The temperature rises to 102° to 104° F., there is intense frontal headache followed by severe backache, and vomiting is usual. Prodromal rashes, either scarlatiniform or purpuric, are occasionally seen, and are chiefly distributed over the lower abdomen and inner aspects of the thighs. The spleen may also be palpable, and a blood count shows a lymphocytosis.

The characteristic eruption appears on the third day, when the temperature falls and the constitutional symptoms clear up. The skin lesions begin as discrete firm red papules, which have a peculiar " shotty " feeling. After three days they become vesicular. Vesiculation begins at the edge of the papule and works towards the centre, and when fully developed the vesicle is multiloculated and may show a central depression or umbilication. The fluid in the vesicles is at first clear, but becomes purulent after about a week. The temperature may again rise at this stage owing to the amount of septic absorption. The pustules may rupture, leaving an ulcer which scabs over, or else the contents may gradually dry up and become converted into scabs, which loosen and are shed during the third and fourth weeks, falling last of all from the palms of the hands and the soles of the feet. The amount of scarring depends upon the extent and depth of the lesions. The prevalent mild type of smallpox leaves practically no scarring, but in the severe type pitted scars remain, and are often most numerous on the face.

The distribution of the rash is important in regard to diagnosis. The papules first develop on the head, face, scalp and wrists—in fact those parts of the skin which are normally exposed. The rash then spreads to the trunk, and finally to the legs. The spots are also most numerous on the face, hands and wrists, feet and ankles, diminishing in number towards the proximal ends of the limbs and on the trunk. The distribution is thus just the opposite to that of chicken pox, and a further difference is that the rash of smallpox comes out quickly, and successive crops of spots do not occur as in chicken pox. Spots may also form inside the mouth, leading to the formation of deep ulcers. The total number of spots varies considerably ; in the mild type there may not be more than half-a-dozen, while a severe case may show several hundreds.

Varieties. In addition to the mild variety which has already been mentioned, two varieties of severe smallpox occur. In the confluent form the spots on the face run together in the pustular stage, making the face so swollen and bloated as to be almost unrecognisable. The lesions on the mucous surfaces may also run together, making the breath fœtid. There is grave toxæmia, and more than half the cases die. In the rare hæmorrhagic form bleeding occurs into the pocks ; the course is then rapid and fatal.

Complications. The milder form of smallpox generally runs an uncomplicated course. In the severe form numerous complications may occur. Broncho-pneumonia is the most dangerous, laryngitis and otitis media may arise, and the eyes may become inflamed, sometimes so severely as to lead to panophthalmitis. Osteomyelitis and nephritis have been recorded. The pustular stage of the eruption may be followed by boils and abscesses in the subcutaneous tissues, with suppuration of the regional lymphatic glands. Encephalitis is also an occasional sequel.

Diagnosis. The diagnosis presents the greatest difficulty in the pre-eruptive stage, and would be impossible without the history of a recent exposure to infection, but if there is this history, then the presence of fever with severe headache and backache should lead to prompt isolation of the patient. It should be remembered that smallpox is peculiar in that a fall of temperature coincides with the appearance of the rash. When the rash has developed it may be mistaken for chicken pox, but a careful study of the distribution will help to prevent error. The vesicles of chicken pox also come out in crops, they are uniloculated, and do not have the " shotty " feel of variola lesions.

Prognosis. Various factors such as the age of the patient, the type of the prevailing epidemic, and the state of vaccination, influence the prognosis. Of these the state of vaccination is the most important. This is illustrated by the epidemic of 1902, in which the mortality among unvaccinated children admitted to the Metropolitan Asylums Board Fever Hospitals was 31 per cent., compared with 1·2 per cent. among the vaccinated children. The good effect of vaccination is particularly seen in children, who, if unvaccinated, carry the highest death rate. Mention has already been made of the increased incidence of smallpox (fortunately of the mild variety) since vaccination has been less strictly enforced.

A severe prodromal stage is not necessarily followed by a severe eruption, but mild prodromal symptoms are an indication that the eruptive stage will also be mild. As a rule one attack of smallpox gives life-long immunity, although second attacks of a mild nature have been recorded.

Treatment. Strict isolation is essential, and children with smallpox are best treated in special smallpox isolation hospitals. Those who have to nurse the patient, as well as those who are contacts, should be given the protection of a further vaccination.

There is no specific treatment. During the febrile period the diet will consist mainly of fluids. For the headache and backache, aspirin or phenacetin can be given, and if the fever is high and there is much restlessness, tepid sponging will be most soothing. During the eruptive stage the mouth should be carefully cleaned with borax before and after each meal. If there is laryngitis, a steam kettle should be employed. The care of the eyes is also important, they should be regularly bathed with boracic lotion, and if the lids are inflamed, a little golden ointment (ung. hyd. ox. flav.) may be gently smeared along their edges.

The irritation of the skin is best relieved by painting the whole body with a saturated solution of potassium permanganate and repeating this each day with a 2 per cent. solution (Rolleston). Alkaline baths may also give relief, and exposure to ultra-violet light during the early stages of the eruption is stated to be beneficial.

Vaccination and Vaccinia

Vaccination consists of conferring immunity to smallpox by the inoculation of cowpox (vaccinia). The history of vaccination dates from 1796, when Dr. Jenner vaccinated a boy with cowpox matter obtained from a milkmaid, and two months later inoculated the boy with smallpox to which he proved immune.

The exact relation between vaccinia and smallpox is not completely understood. Smallpox occurs in epidemics and is highly infectious, while vaccinia never occurs in epidemic form and is only infectious by direct contagion. Nor does vaccination with vaccinia ever give rise to smallpox, although it is possible to give animals vaccinia by injecting them with smallpox material. The minute inclusion bodies described by Paschen [1] in 1926, and found both in vaccine lymph and in the vaccinia lesions of children are generally accepted as the causal agent of vaccinia and smallpox.

The lymph used in vaccination is obtained from a healthy calf which has been vaccinated a week previously. The lymph is preserved in glycerin. In the child the site of vaccination may be either the outer side of the upper arm, or on the outer aspect of the thigh just above the knee ; because of the permanent scar which results, the latter position is preferable provided that adequate care can be taken in the home to prevent contamination from the evacuations. When performing vaccination the skin should be first cleaned with spirit and allowed to dry. A drop of the lymph is then put on the skin, and through it a scratch is made with a cutting-edge needle, not deep enough to draw blood. The lymph should then be rubbed into the scratch and must be allowed to dry before a dressing of clean white lint is applied. An antiseptic dressing should not be used as it may weaken the lymph. It used to be the practice to scarify three or four areas, but now only one is regarded as necessary.

If the vaccination takes, a small papule appears in three days. This slowly enlarges and becomes vesicular, and is at its maximum at the end of a week. By that time the vesicle is umbilicated, the contents are purulent, the surrounding skin is œdematous and red, and the regional lymphatic glands are enlarged and tender. The temperature is raised two or three degrees. During the second week the vesicle dries up into a scab, which separates in the third week leaving a pitted scar. Should the vaccination fail to take, it should be repeated in a month's time, using a fresh batch of lymph.

The first vaccination should be done between the second and sixth month. Younger infants may have obtained some immunity from their mother, and the chances of the vaccination not taking are therefore increased.

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[1] Paschen, E., *Deut. Med. Woch.*, 1926, Vol. 52.

The immunity conferred by one vaccination is not permanent, and re-vaccination should be carried out after an interval of about ten years. The illness of re-vaccination is milder than on the first occasion, and may not leave any permanent scarring, but the immunity lasts a good deal longer.

Although it is open to parents to refuse their consent to vaccination on conscientious grounds, the medical contra-indications are few. It is unwise to vaccinate if an infant is for any reason in an enfeebled state of health, nor should it be done if there is any affection of the skin.

Complications. Considering the large number of vaccinations that are made, complications are certainly uncommon. Secondary infection may occur, and may account for impetigo, erysipelas, or even cellulitis or gangrene, but if proper aseptic precautions are taken these complications should not arise. The use of glycerinated lymph has done away with secondary infection conveyed in the inoculum.

Secondary Vaccinia. If a child scratches and ruptures the vesicle, the fluid which escapes is capable of giving rise to fresh vaccinia pustules. These may appear on any part of the skin, but usually occur near the original vesicle. Secondary vesicles follow the same course as the initial eruption, and are seldom serious unless delicate structures such as the eye are involved.

Generalised Vaccinia. This is a rare complication, occurring in about 1 in 100,000 cases. Towards the end of the first week a crop of papules appears on the face, trunk, and limbs, and passes through the usual stages of vesiculation, pustulation, and desiccation. It is more likely to occur when there is some extensive skin lesion such as eczema, and it may then run a fatal course, but usually the amount of constitutional disturbance is relatively mild, and the child recovers. The condition may be distinguished from smallpox by the lack of prodromal illness, and the history of recent vaccination. *Post-vaccinal encephalitis* is a rare complication which has, however, occurred more frequently in recent years. A more detailed description of it will be found on p. 530.

Mumps (Epidemic Parotitis)

This is a specific infectious disease characterised by tender swelling of one or both parotid glands. The submaxillary and sublingual salivary glands are occasionally involved as well.

Etiology. Mumps usually occurs in epidemics in small isolated communities such as schools, orphanages or barracks. It is rare in infancy, and reaches its maximum incidence during the school years, and affects boys more often than girls. The causal organism is as yet undetermined, but the infection probably passes from patient to patient by droplet infection or by means of fomites.

Mumps has a long incubation period, usually between eighteen and twenty-two days, but it may be as long as a month.

Symptoms. Generally the first symptom to attract attention is the swelling of one or other of the parotid glands, but this may be preceded for a day or two by headache, sickness, or slight soreness of the throat. The swelling occupies the region in front of and below the ear, filling up the hollow behind the jaw and obliterating the angle of the mandible. The swollen gland is firm and tender, and occasionally the overlying skin may be tense and shiny. There is seldom any redness. The act of opening the mouth may be very painful. A little swelling and redness is often to be seen inside the mouth at the opening of the parotid duct. As a rule the swelling of one gland is followed in two or three days by a swelling on the opposite side, giving the face a pale bloated appearance, which is often more noticeable if the observer stands a few paces away from the child. The condition may however remain unilateral. The swelling of one or both submaxillary glands or of the sublingual glands may occur at the same time as the parotitis, and occasionally the swelling is confined to the submaxillary glands while the parotids escape. The parotitis reaches its maximum in two or three days, and subsides in about a week. At first the temperature is raised to about 101° F., but settles down as the glands diminish. The effect on the salivary secretion varies ; in some cases it may be so reduced as to make the throat dry, while in others it may be increased and cause sialorrhœa. Suppuration of the parotids never occurs in uncomplicated mumps, but may arise as the result of a secondary infection. Examination of the blood shows a leucopenia and a relative lymphocytosis.

The outlook is good, complete recovery being the rule. One attack generally confers lasting immunity, but second attacks have been recorded.

Complications. The two most important complications are orchitis and pancreatitis, but fortunately neither are common. Orchitis appears usually towards the end of the first week, although instances have been recorded in which it has preceded the parotitis. As a rule only one testicle is affected, becoming extremely tender and painful, the pain radiating to the lower abdomen, groin and thigh. The temperature is raised, and the amount of constitutional disturbance is likely to exceed that caused by the mumps. The swelling begins to subside after four or five days, but there is a risk of eventual atrophy of the testicle. Inflammation of the ovary may also be a complication, but it is much less common than orchitis.

Acute pancreatitis may also appear towards the end of the first week, but, like orchitis, it has been known to precede the parotitis. The symptoms consist of intense abdominal pain, persistent vomiting, and epigastric tenderness. Although this complication is severe, and must cause much anxiety, recovery without recourse to operation is the rule. During the acute stage sugar may appear in the urine, and subsequent fibrosis of the pancreas may give rise to diabetes.

Of other complications meningitis occasionally arises about the fifth or sixth day, and generally runs a short and mild course. Examination of the cerebro-spinal fluid shows a slight lymphocytosis with increase of protein. Encephalitis is another rare sequel, from which the child usually makes a complete recovery. It does not differ clinically from the encephalitis complicating other infectious fevers such as measles and smallpox. Inflammation of the internal ear may arise, and is serious inasmuch as it is likely to lead to permanent deafness.

Diagnosis. Although mumps is the commonest cause of parotitis in childhood, the gland may become inflamed secondarily to septic conditions within the mouth, particularly during uræmia. There should be little difficulty in distinguishing mumps from acute cervical adenitis if attention is paid to the exact position of the swelling, and even a large mass of swollen lymphatic glands does not give rise to the same difficulty in opening the mouth as occurs in mumps. Chronic swelling of the salivary glands is a feature of Mikulicz's syndrome, but in this condition the lacrymal glands are also affected.

Treatment. There is no specific treatment. The child must be nursed in bed so long as the glands are swollen and the temperature is raised. Owing to the difficulty in opening the mouth, the diet must be mainly of fluids such as milk, broths, and sweetened fruit drinks, and these may be sucked through straws. It is important to keep the inside of the mouth clean ; the mouth should be gently swabbed out with borax or myrrh and honey three or four times a day, and if the child is old enough and can open his mouth wide enough, gargles may be used. Tenderness over the parotids is best relieved by warm fomentations.

If orchitis occurs, the inflamed parts should be supported by a suspensory bandage. Warmth should be applied locally, and small doses of opium may be given by mouth. Pancreatitis should be treated in much the same way by absolute rest, warmth to the epigastrium, and opiates internally.

In the absence of complications, the child may be allowed up as soon as the temperature has fallen and the swelling of the glands has subsided, but he should remain isolated for a fortnight from the onset. Children that have been in contact with a case of mumps should be quarantined for four weeks.

Typhoid Fever

Typhoid fever occurs at all ages, and has even been met with in the newborn, for B. typhosus is one of those organisms capable of transmission through the placenta. Otherwise the mode of infection is the same in children as in adults, namely, by ingestion, the organism being conveyed to the mouth either on the fingers or by infected food and drink. An infected water supply may account for serious epidemics, flies are a proven source of transmission by contaminating food, and children may also become infected by playing about on infected soil round dustbins or cesspools.

The incubation period is about fourteen days.

Symptoms. The symptoms show more variation in children than adults. A typical attack begins gradually with headache, drowsiness or irritability, abdominal pain, loss of appetite, and a rising temperature. Nose bleeding may be an early symptom, or a cough accompanied by signs of bronchitis may occur at the onset. The temperature rises by one degree each evening, remitting in the morning, until by the end of a week it has reached 103° or 104° F. The pulse also rises, but not in proportion to the temperature, indeed the relatively low pulse rate may suggest the correct diagnosis. By the end of the first week the abdomen appears tumid and there is some slight tenderness over it. Palpation may give rise to an unusual amount of gurgling, and on gentle percussion a tympanitic note extends far out into each flank. As a rule the stools are increased in number and become loose and unformed, but occasionally there is constipation at the onset, and rarely this continues throughout the course of the illness.

Towards the end of the first week the spleen may become palpable, but partly because it is soft, and partly because of the abdominal distension and tenderness, it may be very difficult to feel. Its enlargement may, however, be made out by percussion. At this stage also the "rose spots" appear on the skin. These are small pale pink spots scarcely larger than a pin's head, slightly raised, fading on pressure, and occurring chiefly on the front and back of the trunk. Occasionally they are so numerous that they can hardly be overlooked, but more often there are only a few of them, and unless the child is examined in a good light they may easily be missed. Each spot only lasts for two or three days, but as they appear in successive crops the rash may persist for a week or more. A blood examination shows a leucopenia with a relative lymphocytosis.

During the second week the temperature remains at a high level, about 103° F., and the child lies on his back with flushed cheeks, dulled eyes, and an apathetic expression ; there may be delirium ; the tongue, which at first is covered with a whitish fur, becomes dry and brown, the tumidity and tenderness of the abdomen increases, the stools become more frequent, the pulse rises and becomes soft, and there is an obvious loss of flesh.

During the third week, in the absence of complications, the temperature gradually settles by lysis. The child may become constipated, the tongue slowly clears, and the abdominal distension lessens.

The description given above is that of a fairly severe attack. It must be said, however, that typhoid in childhood tends to be a less severe illness than in adults, and not infrequently improvement has already set in at the end of the second week. Nor is the onset always gradual, for it may resemble the onset of pneumonia, with a temperature which rises rapidly to 104° F. and remains at that level. In children vomiting at the onset is not uncommon, and to begin with there may be some stiffness of the neck sufficient to suggest meningitis.

When typhoid occurs in infancy it may easily be mistaken for an acute non-specific entero-colitis. The onset is likely to be brisk with vomiting and high fever, but the course is shorter, generally lasting about a fortnight. As in later years abdominal fulness, an eruption of "rose spots," and a palpable spleen, are to be expected. The disease in infancy carries a higher mortality than in older children.

Diagnosis. In the early stages the diagnosis presents great difficulty, and it is not until rose spots appear or the spleen becomes palpable that the diagnosis is likely to be made.

During the first week the gradually increasing tumidity of the abdomen must always be suggestive, especially if it is accompanied by diarrhœa, for in young children diarrhœa due either to dietetic errors or to non-specific infections is generally accompanied by a hollowing out of the abdomen. The presence of a leucopenia is also of some diagnostic value. The final proof rests on the bacteriological diagnosis. The Widal reaction, which consists of the agglutination of typhoid bacilli by the patient's serum, does not become positive until the end of the first week, and it is generally better to wait until the tenth day before employing the test. The development of agglutinins increases as the disease advances, and the test remains positive for some years after an attack. During the first week typhoid fever is a septicæmic condition and therefore at this stage a blood culture may be positive, or the organism may be cultured from the stools. At about the beginning of the second week the bacilli begin to be excreted in the urine, thus offering a further means of establishing the diagnosis, although by now the Widal reaction offers a quicker and more reliable criterion.

Other conditions may easily be mistaken for typhoid. Tuberculous meningitis may give rise to difficulty, for headache, irritability, fever, vomiting and constipation, and even some stiffness of the neck may occur in both conditions. Abdominal tenderness is however not a feature of meningitis, and the abdomen tends to be retracted rather than full. There may be even greater difficulty in deciding between acute miliary tuberculosis and typhoid. The events leading up to the illness must be most carefully reviewed, and a thorough search of the eye grounds should be made for choroidal tubercles, which if present would put the diagnosis of acute miliary tuberculosis beyond doubt. Tuberculous peritonitis may sometimes give rise to difficulty, but the more gradual onset and the doughy or packed feeling of the abdomen are distinctive features. In infancy the likelihood of typhoid should not be entertained until such common causes of parenteral diarrhœa as pyolitis and otitis media have first been excluded.

Complications. These are fortunately much less common in children than in adults. The two most important are intestinal hæmorrhage and perforation of the intestine, both being most likely during the third week. The first indication that hæmorrhage is taking place is a rapid collapse, the temperature falling below normal while the pulse rate steadily mounts. Provided the child survives long enough, blood will appear in the stools, but death may occur before there is time for this. When perforation takes place there is sudden and severe abdominal pain making the child cry out, the abdomen becomes tense, the temperature falls, and the pulse rate increases. The condition calls for immediate operation, otherwise general peritonitis follows and is almost always fatal. In young infants broncho-pneumonia may occur as a terminal complication, but is rare in older children. Typhoid osteomyelitis and periostitis may arise as a late complication; the long bones are most likely to be affected, usually near the epiphyseal line, and there is a danger of separation of the epiphysis. A complete shedding of hair may occur during convalescence.

Prognosis. The amount of intestinal ulceration is less in children than in adults, and because of this the illness is on the whole milder, hæmorrhage and perforation are less frequent, and the prognosis is correspondingly better. The mortality lies between 5 and 8 per cent. Children show the same liability to relapses as do adults. The relapse appears after the temperature has been normal for a week or so, and runs a shorter and milder course than the original attack. Occasionally there may be more than one relapse.

Treatment. The child must be isolated, and expert nursing is required. Every precaution must be taken to prevent the spread of infection. The urine and fæces should be mixed with 1 in 20 carbolic solution and allowed to stand for two hours before being thrown away. Similarly the child's clothing and bedding should be soaked in 1 in 20 carbolic solution before being laundered. The child must, of course, have his own eating and drinking utensils, which should not leave his room. The attendants must be scrupulous in scrubbing their hands in an antiseptic before leaving the patient's room.

In a long wasting illness such as typhoid, care has to be taken to prevent bedsores by rubbing the back and pressure points with methylated spirit. If constipation is troublesome, the bowels should be moved by a simple enema, never by purgatives given orally. The mouth and tongue should be cleaned with glycerin and borax before and after each meal. During the second and third weeks the temperature and pulse should be recorded every three hours in order that the onset of hæmorrhage may be speedily realised.

Diet. The main article of the diet must be milk, but the diet need not be limited to this. If there is considerable distension of the abdomen it is better to peptonise the milk, and so long as the fever remains high, and also in infancy, it should be diluted with an equal quantity of water. Water and sweetened fruit juices may be given freely. After infancy, foods such as chicken broth, veal broth, custard, junkets, cornflour, groats, and Bengers' may be given to all but the most severe cases. An egg beaten up in milk may also be allowed, and grated plain chocolate in milk is generally welcomed. More solid foods should not be added to the diet until the temperature has remained normal for a week.

There are no specific drugs for the treatment of typhoid fever, and the indications for drugs are few. Intestinal antiseptics do not influence the course of the disease and may irritate the intestine, and so should not be given. Reports indicate that sulphanilamide is likely to prove beneficial, both in the acute disease and in dealing with carriers, but sulphanilylguanidine as an intestinal antiseptic has proved disappointing.

There are special symptoms that may need treatment. Delirium and hyperpyrexia are best dealt with by tepid sponging. A watch must be kept for signs of cardiac weakness such as an increasing tachycardia, softness of the first sound at the apex, or a slight increase in the area of cardiac dullness, and if these occur absolute rest is necessary, and a small dose of digitalis, such as two minims of the tincture to a child of two years of age and upwards, may be given three times a day in combination with ten to thirty drops of brandy in water. The occurrence of hæmorrhage also demands absolute rest, and the diet should be reduced to iced glucose water. Should the child become restless small doses of Dover's powder (2 gr. at five years) should be given. Opium may also be used when diarrhœa is excessive, and is conveniently given in a starch and opium enema.

The child should not be regarded as free from infection until three consecutive bacteriological examinations of the stools taken at three-day intervals have proved negative.

Serotherapy. The investigations of Felix into typhoid antigens has led to the production of an effective anti-serum,[1] which should be given as early as possible in the course of the disease. The dose for children consists of an intramuscular injection on three successive days of from 7 to 20 c.c. according to the age. As with other foreign sera, anaphylaxis may occur if serum has been given previously, and serum sickness may follow in about ten days. The serum is of no use in paratyphoid fever.

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[1] The serum is prepared by the Lister Institute and marketed by Allen and Hanbury. It contains a high titre of antibody against the two important antigens termed respectively V_1 and O.

Prevention. Apart from the methods already described for preventing dissemination of the infection in the sick room, if the child is known to have been in contact with a patient suffering from typhoid fever immediate passive immunity may be afforded by means of anti-typhoid serum. Such immunity would only last for two or three weeks. Active immunity is conferred by the injection of T.A.B. vaccine, and is of obvious value if the child has to travel abroad, but as a means of protecting a child already exposed to infection it is doubtful whether immunity would develop in time. Each c.c. of this vaccine contains 1,000 million B. typhosus and 750 million B. paratyphosus A. and B. Three subcutaneous injections are given at intervals of five days, the first dose being 0·25 c.c. and the two succeeding doses 0·5 c.c.

Paratyphoid Fever

Paratyphoid fever, as the name implies, is a condition closely allied to typhoid fever. Two organisms are responsible, B. Paratyphosus A., and B. Paratyphosus B. Infection with the former is very rare in this country, but the latter is about as common as true typhoid.

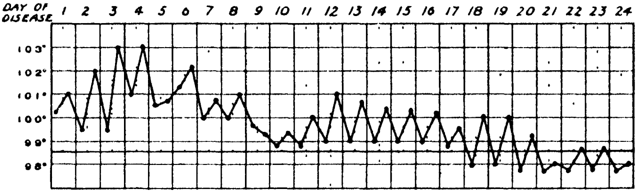

FIG. 133. Temperature chart of an infant aged twenty months, suffering from paratyphoid B. A mild relapse lasted from the eleventh to the twentieth day.

The clinical course of paratyphoid fever closely resembles typhoid, but the illness is less severe and complications are not so likely to occur. The onset may be either gradual or brisk, the temperature reaching 102° or 103° F. By the end of the first week the abdomen becomes tumid and slightly tender, rose spots appear and may sometimes be very numerous, and the spleen may become palpable, but is felt less frequently than in typhoid. The course is also a little shorter, for the temperature generally falls by lysis towards the end of the second week. Blood examination shows a leucopenia. Infection of bone or periosteum may occur as a late complication.

The remarks that have been made concerning the bacteriological diagnosis of typhoid apply also to paratyphoid fever, and in practice the Widal agglutination reaction is always carried out against paratyphoid as well as typhoid organisms. The relative mildness of the illness may give some clinical indication that the child is suffering from paratyphoid rather than from typhoid, but the final differential diagnosis rests on the bacteriological findings.

The prognosis is good, although relapses are just as likely to occur as in typhoid. The treatment is the same as for typhoid fever.

Influenza

This is a highly infectious condition occurring in epidemics, especially in the early months of the year. The epidemics vary in severity, depending chiefly on the varying liability to dangerous respiratory complications, and in magnitude, the most widespread and fatal epidemic being that of 1918. As a rule the illness is less severe in children than in adults.

There is much uncertainty concerning the bacteriology of the disease, largely owing to the difficulty of transferring the infection to animals. This difficulty has recently been overcome by Smith, Andrewes and Laidlaw [1] working with ferrets, and their results point to a filter-passing virus as the primary agent. In 1892 Pfeiffer isolated the bacillus influenzæ from the secretions of patients, and the common association of this organism with the disease has since been amply confirmed, although it remains to show whether the bacillus is more than a secondary invader. The respiratory complications appear to be due to secondary infection by streptococci and pneumococci as much as to Pfeiffer's organism, but the latter has been isolated from the blood, and may be grown in pure culture from the cerebro-spinal fluid in influenzal meningitis.

The disease is conveyed by droplet infection.

The incubation period is short, usually between two and four days.

Symptoms. In its usual form the illness is a mild one. The onset is sudden, and is accompanied by headache, shivering, pains in the back and limbs, cough, and nasal catarrh. There is usually some injection of the fauces. In young infants an initial convulsion, or vomiting and diarrhœa, may occur. The temperature rises rapidly to 102° to 104° F. and the pulse increases in proportion. Sweating is often a feature. After two or three days the temperature settles by lysis, but for a week or more the child remains unexpectedly weak, without appetite, and listless. Mental depression, which is so usual a feature of convalescence in adults, is also common in children during the stage of recovery, and shows itself by their peevishness and readiness to cry, or, should they return too soon to school, by inability to concentrate and mental lethargy. During the period of fever blood examination shows a leucopenia.

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[1] Laidlaw, P. P., *Lancet*, 1935, *i.*, 1118.

Each epidemic of influenza tends to show its own peculiar symptoms, and in the more severe types the symptoms may be mainly respiratory, gastro-intestinal, or nervous.

Respiratory type. This may be most dangerous to life. The initial naso-pharyngeal symptoms are more definite, and the infection quickly spreads down the respiratory passages, causing laryngitis, tracheitis, bronchitis or broncho-pneumonia. The temperature remains high, the voice may be hoarse, cough is frequent and troublesome, and examination may show the usual signs of bronchitis. As the infection progresses towards the smaller bronchioles cyanosis appears, being first noticeable in the finger nails and then the lips, and the respiratory and pulse rates mount. At this stage the amount of prostration and respiratory distress is likely to be more than the signs over the chest would seem to warrant, but considerable significance should be attached to one or more small patches of fine sticky crepitations. In the most severe form confluent broncho-pneumonia may develop. Empyema, acute pulmonary œdema, and pulmonary abscess are rare complications. When recovery takes place, resolution of the lung is always a prolonged affair, and some degree of fibrosis usually results. Bronchiectasis may be the eventual outcome, or in after years the child may suffer from repeated attacks of asthmatic bronchitis.

Infection may also spread from the nasopharynx to the accessory nasal sinuses, and acute otitis media is a frequent complication. Acute nephritis is an occasional complication.

Gastro-intestinal type. In infancy infections of all sorts are likely to be accompanied by diarrhœa and vomiting, and influenza is no exception. The diarrhœa may quickly lead to severe dehydration and toxæmia, and must be combated on the lines indicated in Chapter V. In older children the symptoms of the gastro-intestinal type consist chiefly of abdominal pain and repeated vomiting, and the stools become frequent, loose, and contain mucus. The attack may suggest appendicitis, but the pharyngeal catarrh and the diffuse abdominal tenderness with but little rigidity are points of distinction.

Nervous type. The possibility of convulsions in young infants has already been mentioned, and stiffness of the neck and Kernig's sign may also be present. Usually these symptoms are due to meningismus, and if the cerebro-spinal fluid is examined it will be found to be under pressure but otherwise normal. Older children may complain of severe headache, or may show a muttering delirium. A true influenzal meningitis, with the bacillus influenzæ in the cerebro-spinal fluid, occasionally arises, generally in young children under two years of age, and is almost always fatal after running a course of one to two weeks. Myelitis is a rare complication.

Treatment. Even the mildest cases should be nursed in bed, and isolated from other children in the home. The nasal secretions should be burnt. At the onset a grain of calomel should be given, followed by a saline purge. While the temperature is high simple fluids such as water, lemonade, fruit drinks and diluted milk, all well sweetened, should be allowed in plenty. Gruels and broths may also be given. As soon as the temperature begins to settle the appetite for more nourishing foods should be encouraged. Aspirin (gr. 2½ to 5) may be given to relieve headache, and sodium salicylate in an alkaline diaphoretic mixture is of value. The following prescription may be given every four hours :

℞ Sodii salicylatis gr. iiss.
Potassi citratis gr. v.
Liquoris ammoniæ acetatis m. xv.
Syrupi simplicis m. x.
Aquam ad ʒi.

Special symptoms may require treatment. Delirium is best managed by a tepid sponging followed by 2 to 5 gr. of potassium bromide. Sponging may also be used to reduce a high temperature, and to promote sleep. Half a teaspoonful of sodium bicarbonate or two minims of iodine in a half cup of milk may check vomiting, and in infancy subcutaneous or even intravenous salines may be required to relieve the dehydration caused by diarrhœa. Creosote inhalations are useful in laryngitis and tracheitis, while for broncho-pneumonia the treatment described in Chapter XV. may be followed. Signs of cardiac weakness should be treated by absolute rest, obtained if necessary with bromides, and in an emergency strychnine (gr. $\frac{1}{240}$ at one year, gr. $\frac{1}{80}$ at six years) or ½ c.c. of coramine may be injected subcutaneously. When cyanosis is increasing considerable improvement may follow the use of oxygen, and a small venesection may quickly relieve the embarrassed circulation.

Convalescence needs to be a leisurely affair. Rest in bed should be continued for a few days after the temperature has settled, and gentle massage and tonics are beneficial at this stage. Days in the fresh air or a week or two at the seaside should be arranged if possible, and the child should not go back to school until his health has returned to normal.

Glandular Fever (Infective Mononucleosis)

Glandular fever was first described by Pfeiffer in 1899 but has only been recognised with any frequency in this country since the War, owing largely to the investigations of Tidy.[1] The disease occurs principally among children, although no age is exempt, and the sexes are equally affected. In its usual form it is characterised by a brisk onset with fever and malaise, followed in a few days by enlargement of the cervical glands and other lymphatic structures, and accompanied by a lymphocytic reaction in the blood. Although there can be no doubt that the condition is infective in origin the nature of the virus has not yet been determined. The disease may occur either sporadically or in small epidemics, for if one child of a family is affected the others are likely to show the condition after a week or two. It may also occur in small epidemics in schools.

The incubation period varies from five to fifteen days.

Symptoms. The illness sets in briskly with a rise of temperature from 101° to 103° F. Headache, loss of appetite, and complaint of sore throat are usual, but examination of the throat shows little beyond slight swelling and hyperæmia of the tonsils. After three or four days the cervical glands become enlarged, but instead of the "tonsil gland" at the angle of the jaw being principally affected the glands situated about halfway down the sternomastoid show the greatest swelling, and may become as big as pigeons' eggs. The neck is likely to be slightly stiff but the glands are not as a rule tender; they remain discrete, and show no tendency to suppuration. To begin with, the swelling may only affect one side of the neck, the other side becoming involved later. As a rule the glands in the axillæ and groins also become enlarged, although not to the same extent as the cervical glands; the mesenteric glands may also enlarge sufficiently to make them palpable, while the presence of a spasmodic or brassy cough may indicate swelling of the mediastinal glands. In about half the cases the spleen becomes palpable, extending one or two fingersbreadth below the costal margin. It may also be possible to make out some slight swelling of the liver.

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[1] Tidy, H. L., *Lancet*, 1934, *ii.*, 180 and 236.

In a typical case the blood examination shows an increase in the total white count up to the region of 15,000 to 30,000 per c.mm., about 80 per cent. of the white cells being large lymphocytes.

The glandular swellings subside after a few days, and at the same time the temperature falls, but relapses are very common and are indicated by a fresh swelling of glands and a return of the temperature. Throughout the illness the general health is but little disturbed, but during convalescence the children often appear considerably debilitated, and the glands may remain slightly enlarged for several weeks, making recovery a tedious affair.

The incidence of glandular fever varies considerably, and this seems to depend to a large extent on the variety of symptoms which each clinician is prepared to accept as compatible with the diagnosis. For instance it is said that there may be an actual follicular tonsillitis, or even a membrane over the fauces indistinguishable from a diphtheritic membrane although diphtheria bacilli cannot be cultured from it. In other cases urticarial, erythematous or roseolar eruptions have been described, and Tidy states that examination of the blood may show in some cases an early polynucleosis and in others a leucopenia, and that in short there is no single blood picture typical of the disease. Hæmorrhagic nephritis is an occasional complication, and the Wassermann reaction may sometimes become positive during the acute stage.

Diagnosis. In a condition which may show such a varying clinical picture the diagnosis must often present the greatest difficulty. In its usual form however, typified by fever and slight faucial congestion followed by enlargement of glands and spleen and accompanied by a mononucleosis in the blood, glandular fever may be diagnosed with some assurance, and especially if the condition is giving rise to a small epidemic. As a rule stress should be laid on the mononuclear blood picture in confirming a suspected case. The healthy state of the throat and the fact that the glands principally involved are not those at the angle of the jaw would help to distinguish glandular fever from the much more common follicular tonsillitis, while the exact localisation of the cervical swelling should at the same time prevent confusion with mumps. A distinction from roseola must sometimes be almost impossible; the occipital glands are, however, usually enlarged in roseola and not in glandular fever. Difficulty may also arise in distinguishing the condition from leukæmia, but in the latter the constitutional disturbance is greater and there is also grave anæmia. The rapid onset and the short course will help to differentiate glandular fever from tuberculous adenitis and from lymphadenoma.

Treatment. The outlook is uniformly good, and treatment is largely symptomatic. So long as the temperature is raised the child should be in bed and should be isolated. The bowels should be opened by a dose of calomel followed by a saline purge. During the stage of recovery tonics and preparations containing iron should be prescribed.

Undulant Fever

The occurrence of undulant fever in this country has only recently received much attention. It is now known to be caused by the Brucella Abortus, a common contagious infection in cattle causing abortion. The infection is closely allied to Brucella Melitensis, which has been recognised for some years as the cause of Malta fever. Malta fever is conveyed chiefly by the milk of goats, undulant fever by the milk of cattle, but otherwise the two conditions are identical. The infection in milk is destroyed by pasteurisation, which offers a practical means of prevention.

The condition is more common in adults than in children. Of 250 cases in England and Wales only 9 occurred in children under ten years of age.[1]

The onset is as a rule indefinite, and is characterised by complaints of tiredness, headache, loss of appetite and pains in the limbs. There may also be a sore throat, or perhaps some tenderness over the abdomen, and the spleen can usually be felt.

The name " undulant fever " is derived from the temperature chart, which shows periods of waxing and waning, each lasting about a week. During the periods when the temperature is raised there is likely to be a daily swing over several degrees, running up to 102° to 104° F. There may be heavy sweats accompanying these excursions, but otherwise there is often surprisingly little interference with the general health. The blood picture shows a lymphocytosis. The course varies from a few weeks to six months or more, the average time before the temperature finally settles being about three months.

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[1] Dalrymple-Champneys, Sir W., *Lancet*, 1935, *ii.*, 1449.

Diagnosis. The clinical diagnosis of undulant fever without the aid of laboratory methods can hardly be more than a surmise. The condition should be borne in mind in any child who has recurrent fever associated with a palpable spleen, but in whom the amount of constitutional disturbance remains relatively slight. The diagnosis may be put beyond doubt by an agglutination test, carried out in much the same way as the Widal reaction. A positive result is obtained after the fifth day. Blood culture may also be employed, but is less reliable.

Treatment. There is no specific treatment. Fortunately the mortality is very low, and recovery, although made but gradually, is complete. Owing to the temperature it is necessary to keep the child in bed, but in an illness which continues for such a long time the diet should not be restricted. Aspirin may be given for headache and to relieve the pains in the limbs, and recently good results have been reported following the use of Fouadin,[1] an antimony compound. Vaccine therapy is also worth a trial.

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[1] *Fouadin* (Bayer products) is administered by intramuscular injection on alternate days. For an adult, the first dose is 1·5 c.c., the second 2·5 c.c., succeeding doses being 5 c.c. In children, one-half to two-thirds of the adult dose has been found sufficient, giving five injections in all.

APPENDIX I

BASAL NARCOTICS IN CHILDHOOD

Administered One Hour before Operation

Narcotic	Dose
Avertin (rectal) . .	0·1 gm. per kilogramme body weight
Nembutal (oral) . .	0·6 gr. per stone body weight (maximum 3 gr.)
Paraldehyde (rectal) .	1 drachm per stone body weight.

COMMON DRUGS AND THEIR DOSAGE

THE following table gives a list of common drugs and their appropriate dosage at the various ages of childhood. Drugs given by mouth may be prescribed in these doses three times a day.

Ideally the dose of a drug for a child as compared with the dose for an adult should be based on the proportionate weight, but even this is not an absolutely reliable guide, as some drugs are tolerated relatively better by children than by adults (*e.g.*, chloral, bromides, digitalis, belladonna), while others must be used with particular caution (*e.g.*, opium and its derivatives, strychnine). The custom of working out the dose for a child by multiplying the adult dose by the fraction $\frac{\text{age in years}}{\text{age} + 12}$ is approximately correct for the mineral salts, but not for alkaloids.

Drug.	At 3 months	At 6 months.	At 1 year.	At 3 years.	At 10 years.
Adrenalin (hypoderm), 1 : 1,000	♏ 1	♏ 2	♏ 3	♏ 5	♏ 5
Ammonium carbonate	gr. ¼	gr. ¼	gr. ½	gr. 1	gr. 2
Arsenicalis, liq.	—	—	—	♏ 1	♏ 3
Aspirin	—	—	gr. 1¼	gr. 2½	gr. 5
Atropine sulphate (hypoderm)	gr. 1/400	gr. 1/300	gr. 1/200	gr. 1/150	gr. 1/100
Belladonna, tinct.	♏ 2½	♏ 2½	♏ 5	♏ 5	♏ 10
Calomel	gr. ⅛	gr. ¼	gr. ½	gr. 1	gr. 1–3
Camph. co. tinct.	♏ 2	♏ 2½	♏ 5	♏ 5–10	♏ 10–30
Camphor (hypoderm)	gr. 1/10	gr. ⅛	gr. ¼	gr. ½	gr. ½
Castor oil (as a purge)	♏ 20	♏ 30	♏ 60	dr. 3	oz. ½
Chloral hydrate	gr. ½	gr. ⅔	gr. 1	gr. 3	gr. 7
Chloretone	—	—	gr. 1	gr. 2	gr. 4
Coramine (hypoderm)	½ c c.	½ c.c.	½ c.c.	1 c c.	1 c.c.
Digitalis, tinct.	—	—	♏ 1	♏ 3	♏ 5
Ephedrine hydrochloride	—	gr. ⅛	gr. ⅛	gr. ¼	gr. ½
Ferri et ammon. cit.	gr 1	gr. 3	gr. 3	gr. 5	gr. 5–10
Ferri phos. syr. c̄. quin. et strych.	—	—	—	♏ 3	♏ 10
Ferrous carbonate	—	—	gr 5	gr. 10	—
Heroin (hypoderm)	—	—	—	gr. 1/30	gr. 1/12
Hexamine	gr. ½	gr. 1	gr. 2	gr. 3	gr 5
Hydrargyrum c̄ creta	gr. ¼	gr. ½	gr. 1	gr. 3	—
Ipecacuanha, tinct.	♏ 2	♏ 3	♏ 5	♏ 5	♏ 10
Ipecacuanha co. pulv.	gr. ⅛	gr. ¼	gr. 1	gr. 2½	gr. 5
Luminal	—	—	gr. ⅛	gr. ¼	gr. ½
Milk of magnesia	♏ 15	♏ 30	dr. 1	—	—
Morphia (hypoderm)	—	gr. 1/120	gr. 1/60	gr. 1/24	gr. ⅛
Nux vomica, tinct.	—	♏ ½	♏ 1	♏ 3	♏ 5
Opium, tinct.	♏ ⅛	♏ ¼	♏ 1	♏ 2½	♏ 5
Phenazone (antipyrin)	gr. ¼	gr. ½	gr. 1	gr. 3	gr. 5
Pilocarpine nitrate	—	—	—	gr. 1/32	gr 1/16
Potassium bromide	gr. 1½	gr. 2	gr. 3	gr. 5	gr. 10
Potassium chlorate	—	—	gr. 1	gr. 2	gr. 5
Potassium citrate	gr. 3	gr. 5	gr. 5	gr. 5–15	gr. 10–30
Potassium iodide	gr. ¼	gr. ½	gr. 1	gr. 2	gr. 3
Quinine hydrochloride	—	—	gr. ⅙	gr. ½	gr. 1
Rhubarb in powder	—	gr. ¼	gr. 1	gr. 2	gr. 5
Santonin	—	—	—	gr. 1½	gr. 2
Sodium salicylate (with twice the amount of sod. bic.)	—	—	gr. 2	gr. 3–5	gr. 5–10
Stramonium, tinct.	—	♏ 2½	♏ 5	♏ 5	♏ 10
Strychnine (hypoderm)	gr. 1/800	gr. 1/800	gr. 1/400	gr. 1/200	gr. 1/80
Sulphamilamide group of drugs, see p. 715.					

APPENDIX II

ADMINISTRATION OF SULPHANILAMIDE AND ITS DERIVATIVES

Preparations. Sulphanilamide (Prontosil ; Streptocide ; Proseptasine ; Uleron). Sulphapyridine (M. and B. 693). Sulphathiazole (Thiazamide ; M. and B. 760). Sulphadiazine (Sulphanilamidopyrimidine). Sulphamezathine (Sulphadimethylpyrimidine). Sulphanilylguanidine.

Sulphanilylguanidine is absorbed slowly and incompletely, and is used to produce bacteriostasis in the intestine. Of the remaining preparations, sulphanilamide is used in hæmolytic streptococcal infection, but has been superseded to a great extent by the other members, since they are also effective in pneumococcal, as well as meningococcal, gonococcal, and B. coli infections. Sulphathiazole is also inimical to Staphylococci. It is the author's present opinion that sulphapyridine should be selected against pneumococcal infection, and that sulphathiazole should be used in urinary infections.

Dose. It is essential that the drug should reach a concentration in the blood of from 5 to 10 mgm. per 100 c.c. The various preparations are marketed in tablets containing ½ gm., and the following table shows the amount in tablets which should be given at the various ages of childhood.

Age	Dose reckoned in 0·5 gm. tablets.
0–3 months	½ tablet 6 hourly
3–9 months	½ tablet 4 hourly
9 months–2 years	1 tablet 6 hourly
2–5 years	1 tablet 4 hourly
5–10 years	1½ tablets 4 hourly
over 10 years	2 tablets 4 hourly

In order that the required concentration may be reached as quickly as possible, *the initial dose should be doubled.* Administration must continue through both day and night. Less than a full dose should never be ordered, as inadequate dosage is not only ineffective, but may render the organisms drug-fast, while the liability to toxic symptoms is not appreciably diminished. Under certain circumstances, and notably in meningococcal meningitis, the dose as set out in the table above may be exceeded by doubling each dose for the first forty-eight hours.

Almost always the drug can be given by mouth, but should coma or persistent vomiting make this route impracticable, the soluble sodium salt may be given intravenously or intramuscularly. Intramuscular injection has been followed by local neuritis with permanent paralysis, and care must therefore be taken to avoid the vicinity of large nerves.

Toxicity. Mild toxic symptoms include lassitude, anorexia, and cyanosis. More important are vomiting, due to sulphanilamide gastritis, hæmaturia, leading ultimately to suppression of urine, and rashes. When vomiting occurs in relation to each dose, the drug will have to be stopped entirely by mouth, and it should also be discontinued on the appearance of blood in the urine, or of a rash. The rash is as a rule morbilliform or scarlatiniform ; but an eruption indistinguishable from erythema nodosum may occur. Acute hæmolytic anæmia and agranulocytosis are grave complications, but are fortunately rare.

The drug should not be given in the presence of severe anæmia or leucopenia. Foods that contain sulphur, such as eggs, sulphur containing drugs, and saline cathartics should be avoided so long as sulphanilamides are being given, because they increase the likelihood of cyanosis. It is advantageous to prescribe small doses of alkali with the sulphanilamides.

APPENDIX III

VITAMIN PREPARATIONS

Approximate daily vitamin requirements of infants and young children

	A	B_1	B_2		C	D	E, K, P
			Riboflavin	Nicotinic acid			
Infants	2,500 i u.	80-200 i u. (1 mgm = 320 i u.)	1–3 mgm	10–20 mgm.	20 mgm.	500–800 i u double for premature infants	Not known
Young children	4,000–6,000 i u.				50–100 mgm		

Cod Liver Oil. The content of vitamins A and D varies considerably, but commercial cod liver oil is standardised to contain not less than 600 i.u. of vitamin A and 85 i.u. of vitamin D per gm.

Halibut Liver Oil. The vitamin A content is standardised so that there shall be not less than 30,000 i.u. per gm. The vitamin D content is not standardised, but is roughly 30 times that of cod liver oil.

The following table shows the composition of some proprietary preparations :—

i.u. = international unit.

Sherman unit. There is no i.u. for vitamin B_2 ; the Sherman-Bourquin unit is the amount of the vitamin which when fed for eight weeks to a standard rat induces a gain of 3 gm. per week over and above that made by control rats.

VITAMIN A

Preparation	Description	Makers
Avoleum	Liquid 30,000 i.u. per gm.	B.D.H.
	Capsules, 4,500 i.u. per capsule	,,
Carotene Tabloid	2,000 i u per tabloid	B.W.
Davitamon A	6,000 i.u. per gm.	Organon
,, forte	60,000 i.u. per gm.	,,
Essogen	Capsules 2,000 i.u. per capsule	Trufood
Prepalin	Liquid 72,000 i.u. per c.c.	Glaxo
	Capsules, 24,000 i.u. per capsule	,,
	Ampoules, 100,000 i.u. per ampoule	,,
Vitamin A	Capsules, 33,000 i.u. per capsule	Crookes
,, Injection	100,000 i.u. per c.c.	,,
Vitapex	Liquid, 35,000 i.u. per gm.	Paines & Byrne
	Ampoules, 75,000 i.u. per ampoule	,,

VITAMIN B_1

Bemax	40–50 i.u. per teaspoonful	Vitamins Ltd.
Metatone	12 i.u. per teaspoonful	Parke Davis
Benerva	Tablets, 300 i.u. per tablet	Roche Products
	Ampoules, 600 and 3,000 i.u. per ampoule	,,
Benerva compound	Tablets, 300 i.u. B_1, 1 mg. riboflavin	,,
	15 mg. nicotinic acid per tablet	,,
Berin	Tablets, 300 and 900 i.u. per tablet	Glaxo
	Ampoules, 1,600 and 8,000 i.u. per ampoule	,,
Nicorbin	Tablets, 1 mgm. B_1, 10 mgm. nicotinic acid 25 mg. C per tablet.	,,
Betaxan	Tablets, 300 i.u. per tablet	Bayer
	Ampoules, 1,600 and 8,000 i.u. per ampoule	,,
Crysto-Vibex	Tablets, 300 and 900 i.u. per tablet	Parke Davis
	Ampoules, 1,500 and 7,500 i.u. per ampoule	,,
Davitamon B_1	Tablets, 300 i.u. per tablet	Organon
	Ampoules, 600 and 3,000 i.u. per ampoule	,,

Preparation	Description	Makers
Davitamon B_1 forte	Tablets, 960 i.u. per tablet	Organon
	Ampoules, 8,000 i.u. per ampoule	,,
Planavit B^1	Tablets, 330 and 960 i.u. per tablet	May & Baker
	Ampoules, 3,200 and 8,000 i.u. per ampoule	,,
Thiamin Chloride	Tablets, 300, 960 i.u. per tablet	Abbott
	Ampoules, 3,000 i.u. per ampoule	,,
Cofrou Capsules	5 i.u. B_1, 40 Sherman units B_2 per capsule	,,
Vitamin B_1	Capsules, 300 i.u. per capsule	Crookes
	Ampoules, 1,000 and 5,000 i.u. per ampoule	,,
Vitamin B^1	Tabloids, 330, 960 and 1,600 i.u. per tabloid	B.W.
	Liquid, 660 and 3,000 i.u. per c.c.	,,
Vitamin B_1 (Befortiss)	Tablets, 100 and 300 i.u. per tablet	Vitamins Ltd.
	Ampoules, 1,600, 3,000 and 8,000 i.u. per ampoule	,,
Vitamin B^1 (Pabyrn)	Tablets, 300 and 900 i.u. per tablet	Paines & Byrne
	Ampoules, 1,500 and 7,500 i.u. per ampoule	,,
Vitamin B^1	Tablets, 500 i.u. per tablet	Allen & Hanbury
	Ampoules, 660, 1,600, 8,000 i.u. per	
Vitamin B^1	Tablets, 320 and 960 i.u. per tablet	B.D.H.
	Ampoules, 1,600 and 8,000 i.u. per ampoule	,,

VITAMIN B_2

Preparation	Description	Makers
Riboflavin (Lactoflavin)	Tablets, 1 mgm. per tablet	Roche Products, B.D.H., B.W. Crookes
	Ampoules, 1 mgm. per ampoule	
Riboflavin	Capsules, 400 Sherman units per capsule	Abbott
Nicotinic Acid	Tablets, 50 mgm. per tablet	A. & H., Glaxo, Roche Products
	2 c.c. ampoules, 50 mgm. per ampoule	Abbott, Crookes May & Baker, B.W.
Davitamon P.P.	50 mgm. tablets of nicotinic acid	Organon

VITAMINS B_1 and B_2

Preparation	Description	Makers
Pelonin	Tablets and ampoules, each containing 50 mgm. nicotinic acid	Glaxo
Vitamin B Capsules	Capsules, 150 i.u. B_1, 40 Sherman units B_2 per capsule	Abbott
Yeast Tablets	15 i.u. B_1, 12 Sherman units B_2 per tablet	,,
Marmite	(Unstandardised) Contents about 30 i.u. B_1 and 0·033 mgm. B_2 per gm.	Marmite Food Extract Co.
Pyridoxine (vitamin B_6 : Adermin)	Tablets, 10 mgm. per tablet	B.D.H.
	Ampoules, 50 mgm. per ampoule	,,

VITAMIN C

Tablets of 5, 25, 50 and 100 mgm. Also ampoules of 100 and 500 mgm. Sold under such names as Ascorbic Acid (B. W., A. & H., B.D.H., Vitamins Ltd.), Vitamin C (Parke Davis, Crookes, Lilly, Paines & Byrne), Cantan (Bayer), Celin (Glaxo), Cevitamic Acid (Abbott), Planavit C (May & Baker), Davitamon C (Organon), Redoxon (Roche Products). Fructamin (Paines & Bryne), liquid (0·5 mgm. C.), tablets (15 mgm. C), ampoules (40 mgm. C), also contains vitamin P. Rose Hip Syrup (A. & H.) contains 200 mgm. per ml.

VITAMIN D

Preparation	Description	Makers
Calciferol	"Tabloid" 4,000 and 10,000 i.u. per tabloid	B.W.
	solution, 3,000 i.u. per c.c.	

Preparation	Description	Makers
Radiostol	Liquid, 3,000, 100,000, and 200,000 i.u. per gm.	B.D.H.
	Pellets, 3,000 i.u. per pellet	,,
Calsimil	Tablets, 500 i.u., 5 gm. Ca Na Lactate	,,
Ostelin	Liquid, 5,000 i.u. per c.c.	Glaxo
	Emulsion, 350 i.u. per drachm	,,
	Tablets, 500 i.u. per tablet	,,
Ostelin high potency	Capsules, 100,000 i.u. per capsule	,,
Ostocalcium Tablets	Tablets, 500 i.u. and 7½ grs. CaNa Lactate per tablet	,,
Caldeferrum	Tablets, 500 i.u., 0·125 per Ca, 6 25 mg. Fe per tablet	,,
Colloidal Calcium with Ostelin	Liquid, 5,000 i.u. and 0·5 mgm. Ca per c.c.	,,
Glucodin	Glucose with 30 i.u. per drachm	,,
Davitamon D	Liquid, 5,000 i.u. per c.c.	Organon
,, forte	Liquid, 12,500 i.u. per c.c.	,,
Davitamon D, super forte	300,000 i.u. per c.c.	,,
Calcium with Vitamin D	Liquid, 250 i.u. per teaspoonful or capsule	Crookes
	Injection, 5,000 i.u. per c.c.	,,
Vitamin D (Pabryn)	Ampoules, 15,000 i.u. per ampoule	Paines & Byrne

VITAMINS A and D

Preparation	Description	Makers
Adexolin	Liquid, 12,000 i.u. A, 2,000 i.u. D per gm.	Glaxo
	Capsules, 4,500 i.u. A, 900 i.u. D per capsule	,,
Dekadexolin	60,000 i.u. A, 10,000 i.u. D per ampoule	,,
Minadex Syrup	2,250 i.u. A, 375 i.u. D per teaspoonful	,,
Ostomalt	1,100 i.u. A, 250 i.u. D per teaspoonful	,,
Advita	Capsules, 2,000 i.u. A, 200 i.u. D per capsule	Trufood
Radiostoleum	Liquid, 15,000 i.u. A, 3,000 i.u. D per gm.	B.D.H.
	Capsules, 4,500 i.u. A, 900 i.u. D per capsule	,,
Davitamon A. & D.	Liquid, 6,000 i.u. A, 5,000 i.u. D. per gm.	Organon
	Capsules, 3,000 i.u. A, 2,000 i.u. D per capsule	,,
	Comfits, 1,500 i.u. A and 1,000 i.u. D per comfit	,,
Hepicoleum	Globules, 4,500 i.u. A, 1,000 i.u. D per globule	Eli Lilly
Vitapan	Liquid, 35,000 i.u. A, 7,500 i.u. D per gm.	Paines & Byrne
	Capsules, 3,500 i.u. A, 750 i.u. D per capsule	,,
	Tablets, 1,500 i.u. A, 300 i.u. D per tablet	,,
Halibut Liver Oil	Liquid, 23,000 i.u. A, 1,100 i.u. D per gm.	Crookes
	Capsules, 8,800 i.u. A, 425 i.u. D per capsule	,,
Super D Oil	Liquid, 30,000 i.u. A, 24,000 i.u. D per gm.	,,
	Capsules, 11,000 i.u. A, 9,000 i.u. D per capsule	,,
Halidexol	5,740 i.u. A, 382 i.u. D per teaspoonful	,,
Halycalcyne	5,330 i.u. A, 1,060 i.u. D per capsule	,,
Crookes' Emulsion	2,000 i.u. A, 200 i.u. D per teaspoonful	,,
Halibol	Liquid, 40,000 i.u. A, 6,500 i.u. D per gm.	Allen & Hanbury
	Capsules, 4,500 i.u. A, 900 i.u. D per capsule. Also obtainable in capsules with added calcium	,, ,,
Halibol Malt	300 i.u. A, 40 i.u. D per gm.	,, ,,

Preparation	Description	Makers
Haliverol	Liquid, 27,000 i.u. A, 5,400 i.u. D per gm.	Parke Davis
	Capsules, 4,500 i.u. A, 900 i.u. D per capsule	,,
Nadola	Liquid, 27,000 i.u. A, 2,700 i.u. D per gm.	,,
	Capsules, 4,500 i.u. A, 450 i.u. D. per capsule	,,
Haliver Oil	Capsules, 4,500 i.u. D per capsule	Abbott
Oladal	Liquid, 55,000 i.u. A, 5,500 i.u. D per gm.	,,
	Capsules, 6,000 i.u. A, 600 i.u. D per capsule	,,

COMBINATIONS OF A, B, C and D

Preparation	Description	Makers
Abecedin Tablets	4,500 i.u. A, 50 i.u. B_1, 10 mgm. C, 600 i.u. D. B_2 equal to 2½ gm. fresh brewer's yeast, per tablet, or teaspoonful of emulsion	Napp
	One teaspoonful of Abecedin Emulsion equals one tablet, but has no vitamin B_2	,,
Abidon Capsules	4,500 i.u. A, 30 i.u. B_1, 10 Sherman units B_2, 450 i.u. D per capsule	Parke Davis
Abidon with Vitamin C Capsules	4,500 i.u. A, 50 i.u. B_1, 20 Sherman units B_2, 450 i.u. D, 10 mgm. C per capsule	,,
Crysto-Vibex with Vitamin C	Tablets, 0·5 mgm. B_1, 12·5 mgm. C per tablet	,,
Iradex	Liquid, 825 i.u. A, 9·5 i.u. B_1, 2 Sherman units B_2, 275 i.u. D per drachm	,,
A.B.D. Capsules (Vitetrin).	6,600 i.u. A, 33 i.u. B_1, 0·02 mgm. B_2, 1,320 i.u. D per capsule	Squibb
A.B.D. Capsules	3,000 i.u. A, 100 i.u. B_1, 40 Sherman units B_2, 200 i.u. D per capsule	Allen & Hanbury
Halibol B Capsules	4,500 i.u. A, 10 i.u. B_1, 900 i.u. D, B_2 equal to 5 gr. dried yeast per capsule	
Haliborange	600 i.u. A, 2·5 mgm. C, 150 i.u. D per gm.	,, ,,
A.B.D. Capsules	4,500 i.u. A, 30 i.u. B_1. 10 Sherman units B_2, 450 i.u. D per capsule	Abbott
Penta-Kaps	500 i.u. A, 75 i.u. B_1, 20 Sherman units B_2, 10 mgm. C, 500 i.u. D per capsule	,,
Haliver Malt with Viosterol	3,500 i.u. A, 18 i.u. B_1, 6 Sherman units B_2, 1,000 i.u. D per teaspoonful	,,
Medikaps	Capsules, 6,600 i.u. A, 33 i.u. B_1, 500 i.u. C, 1,320 i.u. D, 22 mgms riboflavin per capsule	Savory & Moore
Multivite	Pellets, 2,500 i.u. A, 160 i.u. B_1, 10 mgm. C, 250 i.u. D per pellet.	B.D.H.
Radiostoleum Emulsion with Vitamin C	1,500 i.u. A, 2·5 mgm. C, 300 i.u. D per gm.	,,
Complevite	Tablets (green) containing 135 i.u. A, 100 i.u. D	Vitamins Ltd.
	Tablets (white) containing 67 i.u. B_1, 133 i.u. C.	,,
Nestrovite	Emulsion, 5,000 i.u. A, 83 i.u. B_1, 15 mgm. C, 500 i.u. D per teaspoonful	Roche Products
	Tablets, 5,000 i.u. A, 166 i.u. B_1, 20 mgm. C, 500 i.u. D per tablet	,,
Davitamon 5	Tablets, 1,000 i.u. A, 50 i.u. B_1, 0·5 mgm. PP factor, 10 mgm. C, 200 i.u. D per tablet	Organon
Homicebrin	3,000 i.u. B_1, 200 Sherman units B_2, 200 i.u. C, 1,000 i.u. D per drachm.	Eli Lilly
Vitamin Quota	Capsules, 4,500 i.u. A, 100 i.u. B_1, 14 Sherman units B_2, 450 i.u. D per capsule	Crookes

Preparation	Description	Makers
Glucose B.D.	75 i.u. B_1, 75 i.u. D per teaspoonful	Crookes
Priovit	Pellets, 1 mgm. B_1, 25 mgm. C, 15 mgm. nicotinic acid per pellet	Bayer

VITAMIN K

Sold under such names as Vitamin K (Crookes), Manaphthone (B.W., Paines and Byrne), Kapilon (Glaxo), Davitamon K (Organon), Prokayvit (B.D.H.), Synkavit (Roche Products), and put up in tablets of 10 mgm., ampoules of 5 and 10 mgm., capsules of 10 mgm., and oil containing 10 mgm. per c.c.

VITAMIN P

Permidin	Tablets, 150 mgm. per tablet	Glaxo
Vitamin P	Ampoules, 10 mgm. per c.c.	Crookes
Vitamins P. and C	Tablets, 10 mgm. C, 10 mgm. P per tablet	,,
	Ampoules, 50 mgm. C, 10 mgm. P per c.c.	,,

In the preparation of this table acknowledgments are due to the manufacturers

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